



# MEDICAL DIAGNOSIS:

## A MANUAL OF CLINICAL METHODS.

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—  
*THIRD EDITION.*

“Felix, qui potuit rerum cognoscere causas.”—VIRG., *Georg.* II. 490.

EDINBURGH:  
BELL & BRADFUTE, 12 BANK STREET.  
LONDON: SIMPKIN, MARSHALL, & CO.

1887.

E06071

EDINBURGH :  
PRINTED BY LORIMER AND GILLIES,  
31 ST. ANDREW SQUARE.

## PREFACE TO THE SECOND EDITION.

THE demand for a Second Edition, thus early, has put it in my power to make certain changes, and to introduce a considerable amount of new matter, which may, I trust, add to the usefulness of the work. While every section has been carefully revised, and in many cases enlarged, the additions will be found mainly in the chapters treating of the general condition of the patient, the symptomatology of the diseases of the Alimentary System, the use of the laryngoscope and ophthalmoscope, and the subject of Electro-diagnosis.

In order to keep the book within moderate compass, the whole chapter on the Reproductive System, which properly belongs to the specialist, has been omitted.

Numerous woodcuts have been introduced to illustrate the text, which it is hoped will prove of assistance to the reader.

J. G. B.

16 AINSLIE PLACE,  
EDINBURGH, *October*, 1883.



## PREFACE TO THE FIRST EDITION.

It is a creditable characteristic of the treatment of disease in the present day that it seeks to proceed on rational principles. Some there may still be who think it enough to give a name to a collective group of symptoms, and treat the disease as they have been told an affection bearing that name should be treated. There may be others who seize upon a few prominent symptoms, and direct their remedies exclusively to these. But every day is, happily, reducing the number of those routine practitioners, and teaching that the true physician is he who seeks thoroughly to investigate the phenomena of disease, that in this way he may the better arrive at a knowledge of that from which they proceed, and to which, therefore, his treatment should be directed. But this can only be arrived at by a thorough knowledge of every change which disease produces in the body, and by a clear conception of what that change imports. This constitutes the science of Diagnosis, and without accurate diagnosis there can be no rational treatment.

The signs and symptoms of disease are changes produced in the animal economy, which are cognoscible by our senses—some by one, others by another; while to assist these senses we call in the aid of instruments which extend their range or increase their power, and of the various analytical processes which the science of Chemistry places at our disposal.

In the following pages an attempt has been made to describe

these signs and symptoms of disease, and to show what is their value from a diagnostic point of view. If this attempt be at all successful, it may enable the student of disease to save much valuable time, by assisting him in analysing and weighing the evidences of disease, and extracting from the whole phenomena which are presented to him those which are of value as indicating its nature. The attempt is made not with the view of checking but rather of encouraging minute inquiry, while it aims at giving to the result of that inquiry more definite form.

A man who has clearly grasped a case in its entirety, who has separated the essential from the accidental, and who has ascertained the weight and bearing of each individual symptom, can go steadily forward in the treatment of his case without experiencing that harassing doubt which arises from partial or crude observation, and which to a conscientious mind, cannot but prove a severe trial.

I desire to express my thanks to my friends who have encouraged and aided me in carrying out my design; among others, to Professor Grainger Stewart, from whom I have uniformly received much kind sympathy and advice. To Professor C. S. Roy I am indebted not only for the heart and pulse tracings with which I have illustrated Chapter XIII. but for very valuable assistance which I have received at his hands. Dr. Alexander R. Coldstream, of Edinburgh, has materially assisted me in the correction of the proof-sheets.

J. G. B.

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## INTRODUCTION.

A PHYSICIAN, when consulted by a patient, is naturally enough expected to be an attentive listener to what, to his informed mind, is a strange medley and most confused account of those deviations from health or actual sufferings by which the patient has been driven to seek aid. The more serious symptoms are often lightly touched upon, the more trivial exaggerated, and the whole jumbled together without logical sequence or the slightest attempt at orderly arrangement. This story, trying as it is to the physician, and all the more trying the more his own mind is duly trained, he ought to listen to ; for this the patient expects, and perhaps has a right to expect. During the tedious narration, it may give him patience to bear in mind two considerations : first, that from it he must obtain the right end of the clue which is to guide him in the difficult task of ascertaining the nature, extent, and seat of the disease ; and second, that by this often most prolix narrative, taken along with his attitude, manner, and expression, the patient absorbed in his own sufferings, is giving his physician, if he is careful and observant, the best opportunity of becoming acquainted with the *ego* with whom he has to deal.

The most critical examination of symptoms, the most careful inquiry into the state of internal organs, the most logical deductions from these as to the morbid changes from which they have originated, will often be erroneous unless the physician is

also a student of human nature, and is able to arrive almost intuitively at some knowledge of the mental characteristics and peculiarities of his patient.

But sooner or later—and more often late than soon—the patient will have arrived at the end of his narration, and then the physician must unravel for himself this tangled web; and, taking the different threads, he must follow them up, and by means of close physical examination, ascertain the condition of the various organs of the body—particularly those which the train of symptoms detailed indicate to be implicated in the morbid process. It is only by a methodical examination of the different systems of the body that a satisfactory view of the condition of the patient can be obtained, and the very foundation of rational treatment laid.

In the following pages an attempt will be made to explain the meaning and diagnostic significance of the chief symptoms and physical signs which are met with in disease. These group themselves naturally round the different physiological systems of the body—Alimentary, Absorbent and Hæmopoietic, Circulatory, Respiratory, Integumentary, Urinary, Nervous, Locomotory; and under those headings they will be considered.\* This is not, of course, to be looked upon as a rigidly accurate division, but for practical purposes it suffices, and it has this great advantage—viz., that those who are habituated to follow such an arrangement in the examination of patients, are less apt to neglect minute points which might otherwise escape the memory. Nor is it to be supposed that every patient requires to be subjected to so exhaustive a catechising as this arrangement, if fully carried out, would necessitate. Many trivial complaints call for no such exercise of patience either on the part of the physician or on that of his patient, and in severe or urgent cases the first examination must necessarily be at best rapid and limited. Nor even where close inquiry is desirable, is it necessary to follow accurately the

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\* See Appendix.

sequence here given ; and to some it may seem more suitable to clear up, first of all, the details regarding that system which seems most profoundly implicated, and only thereafter, and more cursorily, to examine into the condition of the others.

It must be carefully borne in mind that in examining a patient we are dealing with a fellow-creature, and that all our inquiries and all our investigations must be conducted with the utmost courtesy, kindness, and patience.

In the following pages attention will first be directed to certain preliminary inquiries which should be made, and then to the various systems, in the order already mentioned.



## CHAPTER I.

### *The General Aspect, Condition, and Circumstances of a Patient.*

BEFORE entering upon the minute examination of a patient, there are several more general and preliminary inquiries which should be made, and it is needless to say that the care and extent of the investigation required, must depend on two factors: first, on its necessity, in view of the special disease present; and second, on the mental and bodily condition of the patient.

After noting the patient's name, age, occupation, residence, &c., it is well to record, in as brief words as possible, and in his own language, his chief complaint. This is not to be in any sense a statement of diagnosis, but simply the patient's own impression concerning his case. Both in cases of phthisis and bronchitis, for example, we might be told that the patient sought advice on account of severe cough, and of this symptom we would make note as the most prominent in his own mind. We further ascertain, as closely as we can, the duration of the present illness, and record it briefly—so many days, months, or years, as the case may be.

Having thus formed in our minds a general idea, however ill-defined, of the case before us, we proceed to consider the

**Family History.**—Inquiry into the general health of the patient's family should be specially directed to ascertain whether

any of his near relatives have suffered from those forms of disease which are usually supposed to be hereditary, such as consumption, scrofula, syphilis, rheumatism, gout, heart disease, and various nervous disorders. Such inquiry should not limit itself to the near relatives, father, mother, brothers, and sisters, but ought to extend to the uncles, aunts, and grandparents.

### **Habits and General Surroundings at Home and at Work.**

—Luxurious habits, “fast” living, and excesses of all kinds are frequently the cause of disease, and any evidence of these must be sought for, and among them excessive alcoholic indulgence stands out prominently. To defective or unwhole some diet many ailments may be traced, as well as to long hours of work, and to the bad ventilation or defective drainage of the apartments used. Insufficient or ill arranged clothing is also a frequent cause of disease.

It is also well known that certain occupations have a special tendency to produce disease. These may be classed as follows, according as they are due to the following causes :—

(a.) *Mechanical effects of inhalation of dust*, as in knife-grinders, coal miners, and stone masons.

(b.) *Poisonous effects of materials worked in*—such as arsenic, lead, copper, mercury, petroleum, wool containing anthrax, sewer-gas, &c.

(c.) *The position and movements required*.—If certain peculiar movements are very frequently repeated, a condition of spastic muscular contraction is apt to arise, which, when it shows itself in clerks, is known as writer’s cramp, but which is also met with in connection with such occupations as engraving, sewing, type-setting, harp-playing, &c. There is further the position of the body to be considered, whether sitting, standing, or kneeling; and finally, the amount of muscular exertion which the particular employment demands.

**Previous General Health.**—We should endeavour to ascertain the usual state of health, the date and nature of former

ailments, liability to particular morbid conditions, present or previous residences, or other circumstances which may have influenced its production or development, exposure to contagion, &c.; and if the patient be a female, it may be advisable to inquire into the condition of the reproductive functions.

**Origin and Course of the Present Illness.**—It is impossible here to do more than indicate certain general lines on which it is usual to proceed. Having already fixed the date of commencement of the illness, we would next endeavour to gain some accurate idea of the *manner* in which it commenced, with what symptoms, whether it came on suddenly or gradually, to what cause the patient traces his loss of health; and if his statement does not appear to us probable, we must strive, by careful, guarded, and unobtrusive cross-examination, to satisfy ourselves on these points. Knowing the usual etiology of such a case as the one we are studying, we possess a guide as to the direction in which our inquiries should be made. The sequence of symptoms may now be ascertained, the date of origin of each, and its severity; and finally, we note to what medical treatment the patient has been subjected, and what was its result in his case.

### PRESENT CONDITION.

Before proceeding to the examination of each system of the body, it is advisable first to note certain general facts, as follows:

1. Height and weight.
2. Development and muscularity.
3. Condition of the skin as to
  - (a.) Colour.
  - (b.) Perspiration.
4. Condition of the subcutaneous tissue—
  - (a.) Adiposity.
  - (b.) Œdema.
  - (c.) Emphysema.

5. Expression of the face.
6. Temperament, constitution, or diathesis.
7. Attitude.
8. Evidence of previous injury or disease.
9. Temperature.

**1. Height and Weight.**—In almost all diseases the weight becomes diminished, and in the course of treatment the patient should, when it is practicable, be weighed at regular intervals, when a very valuable indication of the progress of the malady will be in our hands. When, however, we have only the result of one weighing, it is of consequence to know what a man of a given height ought to weigh when in health. For this purpose, Mr. Hutchison has compiled a table (deduced from the examination of 3000 persons), from which the following figures are taken, which must, however, not be regarded as more than approximate.

A man of 4 ft. 6 in. to 5 ft. 0 in. ought to weigh about										92·26 lbs.
"	5	"	0	"	5	"	1	"	"	115·52
"	5	"	2	"	5	"	3	"	"	127·86 "
"	5	"	4	"	5	"	5	"	"	139·17 "
"	5	"	6	"	5	"	7	"	"	144·29 "
"	5	"	8	"	5	"	9	"	"	157·76 "
"	5	"	10	"	5	"	11	"	"	170·86 "
"	5	"	11	"	6	"	0	"	"	177·25 "
"	6	"	0	"	.....	"		"	"	218·66 "

**2. Development and Muscularity.**—To be typical of perfect health, the various parts of the body must be accurately proportioned one to another. A moderate amount of adiposity is quite consistent with health, provided that the muscular system is correspondingly developed. Generally, as age advances, the tendency to the deposit of fat increases, and this must be borne in mind. At the same time, its rapid accumulation after fifty years of age is not a symptom of health. Spare people are often the longest lived.

### 3. Condition of the Skin as to Colour.

(a.) *Pallor* is due to defective filling of the capillaries, to deficiency in the quantity of the blood, or of the hæmoglobin it contains. Pallor, consequently, may arise from any condition which prevents the proper assimilation of the food (dyspepsia, &c.), from any interference with the formation of the blood (chlorosis, anæmia, &c.), from any disease leading to loss of blood (hæmorrhage) or its nutritive materials (Bright's disease), or, finally, from any affection of the vascular system interfering with the proper propulsion of the blood (mental emotions, fatty heart, mitral disease, &c.). Paleness of the skin can best be appreciated where the epidermis is thin and the true skin very vascular, as on the ears, cheeks, eyelids, or lips.

(b.) *Redness* of skin beyond the natural tint, first and principally shows itself at those points which have just been mentioned in connection with pallor; but it must be borne in mind that those persons who are, by reason of their occupation, exposed to heat or to the weather, are usually ruddy in complexion. Apart, however, from this cause, redness occurs either as a result of increase of the amount of blood in the body, or of its hæmoglobin (as is seen in "full blooded" plethoric persons), or is due to dilatation of the capillaries. The latter cause accounts for the blushing caused by mental emotion, as well as that following the inhalation of nitrite of amyl; and in a similar way may be explained the redness of the scalp and face in hemicrania, and the general redness of inflammation and of fever.

(c.) *Cyanosis* (κύανος, blue), or blueness of the skin, produced by the accumulation of venous blood, varies much in degree. It is first noticeable on the lips, cheeks, conjunctivæ, ears, and point of the nose and fingers, but may become very general. It is due partly to the retardation of the venous flow, so that the blood remains longer in contact with the tissues, and consequently loses more of its oxygen and takes up a larger quantity of carbonic acid, and partly to deficient oxygenation of the blood in the lungs, so that even when it enters the capillaries

it is more or less venous. The former cause operates in valvular affections of the heart when compensation is lost, the latter in laryngeal and pulmonary affections where the proper aeration of the blood is interfered with. It is in congenital malformations of the heart, particularly in cases of constriction of the pulmonary orifice along with perforation of the ventricular septum, that the complete picture of *morbis cœruleus* is formed. The skin then assumes a livid blue colour, particularly on the nose, lips, ears, and fingers, and these tissues become swollen and indurated owing to the stagnation of blood. The clubbed fingers, with their broad, curved nails, due to this cause, are characteristic of congenital heart affections.\* Cyanosis may also be produced by spasm of the arterioles, as in chilling of the surface. When jaundice exists along with cyanosis (as, for example, in mitral incompetence when the liver is congested), then the yellow icteric hue mixing with the blue gives rise to a distinct green coloration.

(d.) *Jaundice*, or yellow discoloration of the skin, results from the absorption of bile pigment into the circulation. It shows itself first under the conjunctivæ.† In slight cases there is only a very light tinging, but as the absorption of pigment goes on, the skin becomes citron-yellow, then olive green, and it may, finally, in very severe cases, assume a dark brownish green colour. It is usual to divide jaundice into two varieties: —(1.) *Hepatogenic*, or mechanical, which results from obstruction of the gall duct by catarrh, concretions, parasites, pressure of tumours, &c., from compression of the smaller branches of the duct in the liver substance, either from interference with the portal circulation, or in such diseases as cirrhotic, waxy, or fatty liver. (2.) *Hæmogenic*, arising from abnormal conditions of the blood itself. To this class belongs the jaundice which follows the inhalation of chloroform or ether, and possibly that met

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\* Clubbing of the finger ends is also occasionally seen in connection with phthisis.

† The observer must not mistake for jaundice the yellow colour of small masses of fat which may be deposited there in advanced life.

with in typhus, yellow fever, acute yellow atrophy of the liver &c. It appears probable that, in these cases, the jaundice is to be explained by the fact that, owing to the abnormal breaking down of red blood corpuscles, the liver is furnished with an excessive amount of material for bile formation. The result of this is that more bile is formed, the small biliary ducts are over-filled, excretion cannot go on with sufficient rapidity, and absorption takes place.

The internal use of santonin occasionally leads to a yellow discoloration of the skin, but the examination of the urine will at once distinguish this from jaundice.

(e.) *Bronzing* occurs in Addison's disease (disease of the suprarenal capsules). The pigment is deposited in and between the cells of the Rete Malpighi, and is chiefly developed at the points naturally most liable to become darker, and also at the seat of any local irritation. The discoloration commences earliest and becomes deepest on the face, neck, hands, and in the axillæ round the nipples, in the groins, genital regions, and on the abdomen. It also occurs in situations where there has been local irritation, such as that caused by the pressure of garters, or by a recent blister. The mucous membrane of the lips, tongue, and mouth is likewise frequently discolored. The patches of pigmentation are (except those caused by local irritation) arranged symmetrically, and their margins are rarely well defined, but rather shade off imperceptibly. Pigmentation of the skin, closely resembling Addison's disease, is met with in chronic phthisis, and in lymphadenoma and leucocythæmia, and the diagnosis of Addison's disease must not be made from the presence of pigmentation alone, unless such pigmentation as has been described is accompanied with the characteristic constitutional symptoms which that disease presents—viz., asthenia, feeble action of the heart, and small, compressible pulse, gastric irritability, gasping, hiccup, sighing, and breathlessness on exertion.\* Smaller and more sharply defined patches of pig-

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\* The whole subject is very fully and lucidly treated of by Dr. Greenhow; see *Trans. of International Med. Congress*, 1881.

(*f.*) *Grey* discoloration of the skin (*argyria*) occurs after the prolonged use of nitrate of silver. It is most noticeable on those portions of the body which are most exposed to light. From the bluish tinge of cyanosis it can readily be distinguished by the fact that in *argyria* the discoloration does not disappear on pressure, as is the case in the former condition.

**Perspiration.**—Increased perspiration (*hyperidrosis*) may be general or local. In some nervous affections, particularly in hemiplegia, it has been seen to be limited to one lateral half of the body. In very many diseases general *hyperidrosis* may be observed, and furnishes sometimes an indication of considerable importance. Such for example, is the perspiration which occurs in almost all organic diseases in the stage of collapse, and that which follows the crisis of continued fevers (critical sweat). One of the stages of *ague* is that of profuse perspiration, and the night sweats of *phthisis* are among its most distressing symptoms. In acute *rheumatism* the skin is habitually moist, and usually whenever there occurs considerable *dyspnœa*, from whatever cause, it is accompanied by perspiration. It must not be forgotten that strong mental emotions (*fear*) may produce copious sweating.

Local increase of perspiration is usually met with in connection with the *axillæ*, and the palms of the hands and soles of the feet. The sweat accumulating in these regions sometimes gives rise to an excessively disagreeable odour (*bromidrosis*), probably due to the presence of an organised ferment.

Diminution of perspiration is seen locally in many chronic skin affections, and generally in almost all conditions of *pyrexia*, in *diabetes*, and sometimes in the *cirrhotic* form of *Bright's disease*.



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\* The whole subject is very fully and lucidly treated of by Dr. Greenhow; see *Trans. of International Med. Congress*, 1881.

ment are sometimes seen in pregnancy, and in disease of the generative organs in the female; and in *Pityriasis versicolor* the presence of the parasite *Microsporon furfur* gives rise to a brown discoloration.

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Local increase of perspiration is usually met with in connection with the axillæ, and the palms of the hands and soles of the feet. The sweat accumulating in these regions sometimes gives rise to an excessively disagreeable odour (bromidrosis), probably due to the presence of an organised ferment.

Diminution of perspiration is seen locally in many chronic affections, and generally in almost all conditions of diabetes, and sometimes in the cirrhotic form of liver disease.

The perspiration may be coloured yellow from the presence of bile pigments in cases of jaundice, and in uræmia it may hold in solution large quantities of urea. In certain rare cases it has been observed to have a blue colour; and still more rarely extravasation of blood on to the surface of the skin has been observed.

**Condition of the Subcutaneous Tissue.**—There are three pathological states to be noted under this head—viz., (a.) Adiposity; (b.) Œdema; and (c.) Emphysema.

(a.) *Adiposity.*

(a.) *Adiposity or corpulence*, when it passes the limits of health, is a very important condition. It is characterised by the deposit of fat, chiefly in the subcutaneous, the subserous (omentum and mesentery), and intermuscular connecting tissues, and is usually associated either with a plethoric or an anæmic state of the body. The muscles are weak, the heart weak and dilated, the pulse small, rapid, and irregular. Palpitation and dyspnœa occur on exertion, and attacks of *angina pectoris* are not rare. The signs of bronchial catarrh are often present, and perspiration is usually excessive. In very corpulent persons the sexual functions are weak, and sterility is common. These functions appear to be intimately related to the deposition of fat. Where the sexual functions are absent, as in eunuchs and in women after menstruation has ceased, adiposity is common. Sugar is often present in the urine of very corpulent persons. Corpulence is most usually hereditary, but is sometimes brought on independently of this by excessive eating, by the immoderate consumption of alcoholic liquors, or by deficient exercise.

(b.) *Œdema.*

(b.) *Œdema* or anasarca arises from the accumulation of serous fluid in the subcutaneous connective tissue. The swelling usually commences at the ankles—at first appearing only

in the evenings, to disappear after the night's rest—it may increase to a very great extent, the limbs becoming much distended, pale, smooth, and glossy. The finger pressed upon the skin leaves a deep indentation when removed, which only slowly fills up. In well-marked cases the external genitals become greatly swollen. Occasionally the œdema makes its first appearance in the lower eyelid, then usually arising from the acute inflammatory form of Bright's disease. Œdema may arise—

1. From obstruction to the return of venous blood to the heart. In this way it may occur in almost all diseases of the heart, and in some pulmonary affections, particularly emphysema. Pressure on the inferior vena cava in the abdomen, from an enlarged liver or tumour of any kind, or on the veins of the leg, may give rise to œdema; and local œdema is common round abscesses.

2. From alteration in the coats of the vessels, arising from a blood supply defective in amount or in quality. Under this heading fall the so-called hydræmic dropsies seen in diseases of the kidneys, in anæmia, and in many wasting diseases.

In *myxœdema*, a rare disease recently described by Dr. Ord, the subcutaneous tissues are infiltrated with a peculiar mucus-yielding substance, which causes a dropsy-like swelling all over the body. The face is most characteristic. It is swollen, waxy, and expressionless, the swelling differing from ordinary œdema in two important points—viz., first, in not selecting the more dependent parts of the face (such as the lower in preference to the upper eyelid), but invading every feature alike; and second, in its resiliency, the pressure of the finger leaving no subsequent mark. Along with this peculiar œdema there is great hebetude of the nervous system, and in late stages insanity is liable to develop.

(c) *Subcutaneous Emphysema.*

(c.) *Subcutaneous Emphysema* of the skin is produced by a collection of gaseous fluid in the subcutaneous cellular tissue.

The skin pits very slightly on pressure, and there is a curious and unmistakable feeling of crackling under the finger. Apart from surgical causes, emphysema occurs as the result of loss of continuity in the air passages or at some portion of the alimentary tract. Ulceration or wound of the larynx or trachea may give rise to emphysema, but it more commonly results from rupture of one or more air vesicles in the lung. In the latter case, the air is effused into the interlobular septa, and under the visceral pleura passes back to the root of the lung, thence to the mediastinal cellular tissue, and finally appears in the jugular fossa, and passes under the skin of neck and chest. Perforation of the œsophagus, from whatever cause, is liable to occasion subcutaneous emphysema, and ulceration of stomach or intestines may likewise allow of the escape of air under the skin if these structures have been previously glued to the abdominal walls by adhesions.

**The Expression of the Face** is often characteristic of the disease under which the patient is labouring, and is a very valuable indication, especially in children. It is the general effect upon the observer of a combination of a number of traits, such as the colour, volume, and muscular state of the face, the state of the eyes, &c. To some of these allusion has already been made; a few remarks, however, may be added. In connection with the eyes, an injected condition of the conjunctiva is common in febrile conditions (particularly in typhus), and in neuralgia of the ophthalmic branch of the fifth nerve. In chlorosis and anæmia generally, the sclerotic has a peculiar white pearl-like lustre. Prominent eyeballs are met with in cases of exophthalmic gôitre, and in connection with tumours of the orbit. Sunken eyeballs are seen in all wasting disorders, due to the atrophy of the fat which lies in the posterior part of the orbit. In regard to the muscles of the face, we may meet with spasmodic contraction in tic-douloureux, tetanus, hysteria, epilepsy, and chorea. In facial paralysis the muscles are paralysed, and the expression on the diseased side is therefore

lost. While many diseases have expressions of the face which are more or less characteristic of them, there are one or two facies which stand out more prominently than the rest, and which deserve special attention.

*The Typhoid Facies*, that, namely, which is met with in the typhoid or adynamic state, is characterised by the following symptoms:—The patient lies on his back, dull, expressionless, and somnolent, his eyes half shut, with the pupils dilated. The face is usually emaciated, the lips black at the edges and trembling, and wide enough apart to show the teeth, which are covered with sordes.

*Facies of Heart Disease*.—In mitral disease, after compensation has been lost, the face is swollen and pale, except the lips and cheeks which are blue, the veins of the neck are engorged, and the mouth is usually half open on account of the dyspnœa. In aortic insufficiency, on the other hand, the chief characteristic is extreme pallor.

*Facies of Inspiratory Dyspnœa*.—Eyes wide, head thrown back, nostrils dilated but not working, mouth half open, face pale. Seen in croup, œdema glottidis, paralysis of the posterior cricoarytenoid muscles, &c.

*Facies of Expiratory Dyspnœa*.—The face is swollen, dark reddish blue, nostrils working powerfully, the eyes injected, and the mouth open. The patient is sitting up with the arms fixed to some support so as to allow the extraordinary muscles of respiration to come into play.

*Facies of Facial Paralysis*.—The unopposed muscles on the side not affected draw the features over towards that side, so that the face has a curious one-sided appearance, which is most noticeable when the patient laughs or speaks. On the affected side of the face the cheek hangs loose, and puffs in and out with each respiration, the saliva trickles from the corner of the mouth, the eye is open and watery, and the whole skin of that side becomes smoothed out and loses its wrinkles.

*Facies of Bulbar Paralysis*.—The face is expressionless, does not move in laughter and crying, the mouth is enlarged trans-

versely, and saliva trickles out of the corners. The naso-labial furrows are well defined.

*Facies of Hectic Fever* is chiefly characterised by a circumscribed flush on the malar bones. This flush is also seen in acute pneumonia, and is said to occur chiefly on the same side as the disease.

*Facies of Cholera*.—In the stage of collapse of cholera the face is sunken and wrinkled, the eyeballs retracted, and the countenance livid.

*Facies of Acute Peritonitis*.—The face is haggard, the expression distressed and anxious. The upper lip is drawn upwards so as to expose the teeth.

*Hippocratic Facies* is the name given to the expression of the face immediately preceding death. The face is pale and livid, the eyes sunken and lustreless, the eyelids separated, the nose sharp and pinched, and the lower jaw falling.

**Temperament, Constitution, or Diathesis.**—Many persons show in their general appearance that they have a constitution which is liable to certain forms of disease. The recognition of such peculiarities of appearance may be of great importance in cases in which it is difficult or impossible to obtain satisfactory information regarding the family history. The more important of these varieties will here be sketched in outline.\*

1. *The Sanguine Constitution*.—Body well developed, head large, teeth massive and good, complexion ruddy, hair thick, digestion and nutrition good, pulse hard, blood pressure high. In later years the body becomes corpulent, and the signs of old age come on prematurely. Such persons are liable to arthritic affections of all kinds, and to diseases of the heart and blood vessels (angina, fatty heart, aneurism, atheroma, apoplexy, &c.).

2. *The Nervous Constitution*.—Figure small and wiry, face

\* For this outline I am indebted to the courtesy of my friend Professor Grainger Stewart, from whose unpublished lectures it has been in part derived.

mobile, features small and delicate, great activity of mind and body, dyspeptic, and with highly strung nervous system. Individuals of this temperament are specially liable to nervous diseases of all kinds.

3. *Strumous Constitution*.—The whole osseous system badly developed, joints enlarged, mucous membranes irritable, upper lip and alæ nasi thick, thorax contracted, skin pale and delicate, and hair thin. Such persons are liable to diseases of bones, lymphatic glands, and to tubercle in all its forms.

4. *Lymphatic Constitution*.—Body large and clumsy, muscles flaccid, face pale and expressionless, movements slow, and functions both of body and mind sluggish.

5. *Bilious Constitution*.—Face oval, long upper lip and chin, long nose, complexion dusky, digestion sluggish, liver in particular being inactive, nervous system not highly strung. In its further developments, this constitution passes into the melancholic, where the patient takes gloomy views of things in general. Such persons are liable to melancholic insanity.

6. *Gouty Constitution*.—Hair early grey, little tendency to baldness; nose short, rounded, and red; cheeks ruddy, eyes generally dark, teeth large and covered with thick enamel, digestion bad, suffers much from dyspepsia, heart tends to degenerative changes, arteries atheromatous, arcus senilis appears early, and is well marked. Persons of this constitution are liable to all the forms of gout, to the cirrhotic form of Bright's disease, to neuralgia, and to apoplexy.

7. *Rheumatic Constitution* closely resembles the sanguine, as already described. There is, however, in it a greater tendency to fulness of body and less general vigour. The teeth are liable to early decay.

To the other constitutions, such as the malarious, the hæmorrhagic, and the alcoholic, the limit of these pages does not allow allusion, nor need the numerous mixed forms be specially noticed.

**Attitude**.—The attitude of the patient is very frequently



determined by the disease from which he suffers. For example, at the height of a severe fever, the patient may be seen lying on the back very flat, with the face turned upwards; and one of the first indications of improvement, is his wish to be turned on to his side. The round-shouldered appearance of the asthmatic and emphysematous is characteristic; and in bed those who suffer from dyspnœa are usually obliged to be propped up. Again, a patient suffering from acute peritonitis lies with his knees drawn up; and so with all diseased conditions, almost every one of which compels the assumption of some more or less characteristic attitude.

**Evidence of Previous Injury or Disease.**—Apart from the obvious traces of such surgical affections as fractures, &c., it is always of importance to note any indications of previous disease which may meet us in the course of our examination. The pitting of small-pox, the cicatrices over scrofulous glands, the large joints of those who in their childhood have been subject to rickets, old-standing paralysis, are among these indications. The most important are noted in the following paragraph :—

**Cachexia** expresses a chronic condition of ill-health, the result of disease, such as is produced by cancer, syphilis, tuberculosis, &c. The most prominent varieties of cachexia are the following :—

*Cancerous Cachexia.*—There is great emaciation and loss of strength, accompanied with a peculiar dirty, waxy, yellowish, or brownish-green colour of the skin.

*Splenic Cachexia*, that of Adenia or Hodgkin's disease, shows itself by the presence of emaciation, pallor, and weakness, accompanying or succeeding the enlargement of the lymphatic glands throughout the body, along with dropsy and hæmorrhages.

*Scorbutic Cachexia.* — Lassitude, oppression, palpitation, cyanosis, and hæmorrhage from the gums accompanying scurvy ;

the eyes are sunken and surrounded with dark rings, and the skin earthy in colour.

*Syphilitic Cachexia.*—Acquired syphilis in its latter stages is often accompanied with emaciation, weakness, and anæmia; but this is not by any means always the case. Inherited syphilis shows itself in adult life by the following signs. The forehead is prominent, the bridge of the nose sunken (owing to disease of the nasal bones), the cornea is often marked with opacities, the result of inflammation, the iris is sometimes adherent, the skin of the face has an earthy tint, there are traces of fissures at the angles of the mouth, the teeth are pegged (see page 25), scars of ulcers may be found on the palate or over the body, indications of old or of recent disease of bone may show themselves, and nodes may appear on the skull or tibiæ or at some other point. Accompanying these signs there is a general stuntedness of growth, and low vitality, falling out of the hair, &c.

The characteristics of the other cachexiæ (tubercular, gouty, &c.) will be noted in connection with each organ of the body involved.

**Temperature.**—The thermometer used for estimating the temperature of the body ought to be of such a size, and so divided, that tenths of a degree can easily be read off; it ought to have an arrangement for maximum registration; and the purchaser of a clinical thermometer should see that the accuracy of the instrument in question has been tested by comparison with a standard thermometer, which can be done by sending the instrument to the Kew Observatory.\*

It is most usual to take the temperature by placing the instrument (the index having been first jerked down below the normal point) in the axilla, the skin of which region must

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\* It is well known that a thermometer originally correct loses its accuracy after a certain time, owing probably to molecular changes in the glass. Clinical thermometers ought therefore to be compared with the standard from time to time, at intervals say of two years.

be carefully dried, should there have been any perspiration. To give reliable readings, the thermometer ought to remain in position for fifteen to twenty minutes, and during the last five minutes its height should be several times examined, in order to see if it remains stationary, or, not having reached its highest point, is still rising. In the latter case, it ought to be allowed to remain longer, until no further elevation takes place. Still more accurate results are to be obtained, and in shorter time, when the instrument is passed into the rectum. In children the temperature is most readily taken in the mouth, the bulb of the instrument lying under the edge of the tongue.

In all cases of importance the temperature ought to be taken twice a-day, morning (9 to 11 A.M.) and evening (5 to 7 P.M.), and in serious cases every two hours. From the data so obtained, charts should be constructed in the usual manner.

While the *mean normal temperature* may be taken to be 98·6° of Fahrenheit's scale, yet variations from 97·5° to 99·5° are met with in healthy persons. When the mercury stands below the former point, however, it usually indicates a state of collapse. Above 99·5° the thermometer indicates a condition of fever, which, when it surpasses 105°, is spoken of as hyperpyrexia. More important for diagnosis than the actual reading is the manner in which the temperature rises. Four types of temperature curves are to be distinguished.

1. *Continued*.—The temperature rises rapidly and almost continuously to a given height, at which it remains during the time the fever lasts, subject only to such slight differences between morning and evening temperature as may be observed in health, and then it falls to, or even below, the normal point, as suddenly as it rose. This rapid fall, which is coincident with the commencement of convalescence, is denominated *crisis*. This type is met with in typhus, small-pox, measles, &c., but is perhaps best marked in acute lobar pneumonia.

2. *Remittent*.—In this variety the difference between the morning and evening temperatures is considerable (say 2° F.

or more). Hectic fever in all its forms is perhaps the best example, and it is to be noted that occasionally (especially in phthisis) the ordinary state of matters is reversed, the morning temperature being high, and the evening low. During the first few days of typhoid fever the temperature rises in a characteristic manner, each morning remission being considerably under the temperature of the preceding night, but above that of the preceding morning. In typhus fever, as a rule, the rise is more sudden. In well-marked examples of this type of fever temperature, when the disease takes a favourable turn the re-establishment of the normal temperature takes place very gradually, and the slow fall of temperature is called *lysis*, to distinguish it from the more rapid crisis of which we have already spoken.

3. *Intermittent*.—This type is seen in the different forms of ague. The temperature rises with great rapidity to a very high point, and falls again in a few hours to normal. There then follows a period of intermission, when the condition of the patient is normal, and then again the temperature rises as before, and so on. The intervals vary in duration according to the type of ague present, lasting twenty-four hours in quotidian, forty-eight in tertian, and seventy-two in quartan ague.

4. *Relapsing or Recurrent Type*.—After a rigor the temperature rises to a high point, near which it remains for some days, and then falls to normal (crisis). An interval of immunity from fever of varied duration (5 to 8 days or longer) succeeds this attack, and then follows a relapse, which corresponds very closely to the original attack. These relapses may occur several times before the disease ceases.

## CHAPTER II.

### Alimentary System.

THE signs and symptoms connected with the alimentary system may be conveniently considered under two sections. The first of these contains the various objective phenomena which present themselves during the examination of the organs of mastication and deglutition, the subjective sensations of digestion, and the phenomena connected with the expulsion of the food from the body, either by vomiting or by defæcation.

The second group includes all these physical signs which the examination of the abdomen affords.

The first of these sub-divisions will be treated of in the present chapter.

**Lips.**—In examining the lips, we have to note—

(a.) *Colour.*—Owing to the great transparency of the labial epithelium, any change in the colour of the blood circulating in the minute vessels underlying it can be readily distinguished. When the lips assume a dusky-blue cyanotic colour, we know that the circulation is being carried on imperfectly, which may be due to defective circulation locally or in the lungs, or to interference with the entrance or exit of air to or from the pulmonary alveoli, or to a combination of these causes. The lips may have a pale waxy colour, indicating that the amount of hæmoglobin is either absolutely less than normal, as in cases

of loss of blood, or is both absolutely and relatively (in relation to the aqueous constituents of the blood) below the normal standard, as, for example, in chlorosis and other forms of anæmia, as well as in many cases of Bright's disease.

(b.) *Form.*—Abnormal thickness or thinness of the lips gives an indication of the amount of serum contained in the interstices of the tissues. The thin pinched lips which are seen, for example, in the second stage of cholera, indicate an abnormal diminution of interstitial lymph. Herpetic eruptions upon the lips (herpes labialis) occur in feverish conditions, particularly in pneumonia; and in syphilis deep and painful fissures are often met with. When we find the lips dry, cracked, and coated with sordes, as in most febrile conditions, we know that the patient has been breathing through the mouth, this causing (in combination with the raised temperature) an abnormally rapid evaporation of the saliva which in health keeps the lips moist.\* The result of this rapid evaporation is, that the solid constituents of the saliva are deposited on the lips in the form of sordes, whilst the cracking is due to the unequal contraction of the epithelial layer in the act of drying. The fact of the mouth being kept constantly open for the purpose of respiration, although frequently due to more or less complete obstruction of the nasal passages, may also result from any cause which interferes with the proper oxygenation of the blood; the natural tendency in such cases being to attempt to increase the quantity of air respired. All dyspnoëic patients, therefore, tend to breathe through the mouth, as offering a freer passage for the ingress of air than the nostrils. The fur on the lips occurs under similar conditions as does that upon the tongue, which will be presently described in detail.

(c.) *Movements.*—The lips being among the principal organs of expression of the emotions and the will, many affections of the central nervous system lead to trembling of the lips (as for example, delirium tremens). Abnormal contraction and relaxa-

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\* The amount of saliva secreted is also diminished in fever.

tion of the labial muscles usually mimics and exaggerates some normal expression. The *risus sardonius* of tetanus and of strychnine poisoning may be taken as examples of this. The opposite condition is seen in the expressionless appearance which occurs in double facial paralysis.

**Teeth.**—The following tables show the periods at which the various teeth usually appear. It is important in treating disease in children to bear these dates in mind.

#### MILK TEETH.

	APPEAR.	FALL OUT.
Central incisors, under, .	6th month,	7th year.
"    "    upper, .	10th " }	7½ " "
Lateral incisors, under, .	16th " }	8th " "
"    "    upper, .	20th " }	
Canines, under, .	30th " }	12th " "
"    upper, .	32nd " }	
First molars, under, .	24th " "	10th " "
"    "    upper, .	26th " "	10½ " "
Second molars, under, .	28th " "	11th " "
"    upper, .	30th " "	11½ " "

#### PERMANENT TEETH.

	APPEAR.
Central incisors, . . . . .	7th year.
Lateral incisors, . . . . .	8th-8½ " "
Canines, . . . . .	11th-12th " "
Anterior bicuspid, . . . . .	9th-10th " "
Posterior " . . . . .	11th " "
First molars, . . . . .	5th-6th " "
Second " . . . . .	12th-13th " "
Third " . . . . .	18th-25th " "

These periods of dentition are liable to considerable variation even in health. Sometimes the appearance of the milk teeth is greatly delayed in healthy infants, but it should be remembered that very frequently delayed dentition is the result of rickets, and in such cases further evidence of that disease

should always be carefully sought for when teeth are late in appearing.

It is important to observe the *shape* of the teeth. In congenital syphilis the permanent central incisors, usually of the upper jaw, are very often considerably altered, as was originally pointed out by Hutchinson (see Fig. 1). They are shorter and narrower than natural, peg-shaped, with a crescentic notch at the free edge, sometimes grooved down the centre, and set at greater intervals in the gum than is natural.

*Caries* of the teeth must be noted as bearing upon neuralgia, dyspepsia, &c. In young and otherwise healthy subjects, the usual cause of extensive caries of the teeth is acidity of the buccal saliva, arising from a slight catarrhal condition of the

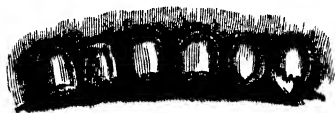


FIG. 1.—Shape of Teeth in Congenital Syphilis.  
(After Hutchinson.)

mucous membrane of the mouth. The continued and excessive use of mercury gives rise to a looseness of the teeth.

**Gums and Mucous Membrane of the Cheeks.**—Like the lips, the gums show by an anæmic, bloodless appearance the presence of defective circulation, or diminution of hæmoglobin in the blood. In chronic lead-poisoning a *blue line* forms on the gums close to the dental margin, caused, in all probability, by deposit of sulphide of lead, precipitated there by the sulphuretted hydrogen generated by the decomposition of particles of food remaining about the teeth. Swelling and tenderness of the gums, along with the looseness of the teeth already noted, are among the earliest signs of the action of mercury. Spongy gums which readily bleed are met with in cases of scurvy. Inflammation of the gums is either general (stomatitis), which may be of parasitic origin (muguet or thrush), or local, constituting gum-boil. Bleeding may also



result from the sharp angles of carious teeth, or from the coming away of tartar from the teeth, causing laceration of the neighbouring soft parts. In paralysis of the buccinator muscle the cheek hangs loose, and the food collects between it and the teeth, This condition is seen in facial and bulbar paralysis.

**Tongue.**—In examining the tongue three distinct points have to be considered—(1) its size and shape, (2) its movements, (3) the condition of its surface.

(1.) *Form.*—In health, the tongue varies much in shape, and this without any particular significance. It becomes swollen from various causes, particularly inflammation, the result of small-pox or scarlatina, or the abuse of mercury or other drugs, and from the presence of cancerous or syphilitic new formations. In dyspepsia the tongue has frequently a swollen sodden character, marked at the edges by the teeth against which it has been pressed and even without these causes where there is great mental hebetude along with defective movement of the organ in the mouth, such œdematous swellings take place. The greatest degree of swelling is however found in cases of acute glossitis.

(2.) *Movements.*—The tongue receives its motor innervation through the hypoglossal nerve, and so long as that nerve and the muscular fibres of the tongue are normal, protrusion of the organ is effected with exactitude and rapidity. When, however, the cerebral functions are in more or less abeyance, as in typhus and other severe diseases, the tongue is only protruded slowly, with difficulty and with tremor. A tremulous tongue is also met with in drunkards, in cases of general paralysis of the insane, bulbar paralysis and progressive muscular atrophy.

Paralysis of the tongue is frequent in cerebral affections (hemiplegia from hæmorrhage, embolism, &c. ; bulbar paralysis, general paralysis, the advanced stages of locomotor ataxia, &c.). In unilateral paralysis the tongue when protruded is inclined towards the diseased side. When the paralysis is bilateral, the

tongue is relaxed, wrinkled, fibrillary contractions may be seen on its surface, and the speech becomes inarticulate and unintelligible.

In chorea, hysteria, and eclampsia, spasm of the lingual muscles is often met with. The peculiar quick spasmodic manner in which the organ is protruded by the choric patient must be seen to be understood, and once witnessed will never be forgotten.

(3.) *Surface of the Tongue.*—Whenever a patient breathes habitually through the mouth\* there is a tendency to dryness of the tongue, as well as of the lips (as has been already stated), because of the more rapid evaporation of the saliva and buccal mucus, which usually keep these parts moist. In fever this is still more marked, partly because there is then diminished secretion of these fluids, and partly because the evaporation goes on more quickly on account of the elevation of the temperature. In order to prevent this dryness, the patient moistens his lips with water at very short intervals, and hence the dry tongue is not always present even in fever. When it is present in such patients, it shows that thirst is not being felt, and is an indication that the senses are becoming blunted, and consciousness is being lost. When the tongue becomes very dry, cracks appear on its surface, due to the unequal contraction of the epithelium in the act of drying. A degree of dryness of the tongue is likewise met with in diabetes, and after the administration of certain drugs—atropine, for example.

*Fur on the Tongue.*—In fever when, as has just been said, the saliva evaporates quickly, it deposits its solid constituents upon the lips and tongue. When, however, no abnormal evaporation is taking place, the fur which is found upon the tongue is not to any appreciable extent so formed. It then consists of *debris* of food, of cast-off epithelial cells, and of

\* Either on account of obstruction of the nasal passages or from anything which interferes with the proper oxygenation of the blood—dyspnoea in all its forms.

masses of micro-organisms.\* These organisms enter the mouth in the air or in the food, are caught on the filiform papillæ, to which they firmly adhere, and there multiply with great rapidity. The fungiform papillæ are too smooth to afford points of attachment, and hence they are usually free from fur, and stand out red and prominent. In scarlet fever these papillæ are more than usually conspicuous, on account of the congestion of the mucous membrane, and they stand out distinctly through

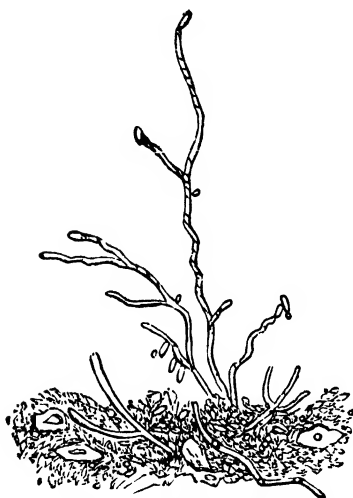


FIG. 2.—*Oidium Albicans*. (Quain's Dict.)

the creamy fur which covers the rest of the organ, and hence the tongue assumes a "strawberry" appearance. The fur which collects on the lingual surface is being constantly detached by the rubbing of the tongue against the roof of the mouth, gums, and teeth; and as during sleep there is very little movement, the coating is always thickest in the morning. It is also to be remarked that the fur collects most where the tongue is roughest, and where the movement is least in amount—*i.e.*, the

centre and back part. Increase of fur may result from any condition which diminishes the movements of the tongue on the palate and gums, such as dryness of the mouth, swelling of the tongue, defect of the palate, or disease of the central nervous system (hemiplegia, bulbar paralysis, &c.) paralysing the lingual muscles.

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\* According to Butlin's cultivation experiments (St. Bartholomew's Hosp. Rep. xv.) these include: *Micrococcus*, *Bacillus subtilis*, *Sarcina ventriculi*, *Spirochæte Obermeyerii*, *Bacterium termo*, and a form of *Vibrio*.

But in addition to the fur which adheres to the tongue, the cavity of the mouth often affords a nidus for other forms of vegetable organisms, such as those which give rise to lactic and butyric fermentation of sugar, and acetous of wine. By far the most important of these is the parasite which occasions thrush, the so-called *Oidium albicans*, which Grawitz has lately shown\* to be identical with the *Microderma vini*. Thrush is usually met with in delicate children, particularly in those who are brought up on cow-milk, and affects the inner surface of the lips, and the mucous membrane of the mouth generally. Small white points first form, which rapidly increase in number, and extend in area, the patches appearing like curdled milk. The reaction of the saliva becomes strongly acid, and thus causes great irritation of the mucous membrane of the mouth, which becomes red, swollen, and painful. When these patches are examined microscopically (see fig. 2) we find, in the midst of a mass of epithelium and salivary corpuscles, the fungus, in the shape of long branching transparent filaments with a double contour, and bearing the spores in a globular receptacle at their extremity. In the meshes of the net-work which these filaments form, there are usually to be seen numerous free spores, which are spherical, and highly refractive. In doubtful cases, the diagnosis of thrush may thus be made by means of the microscope.

The colour of the tongue varies considerably under different conditions. In fever it is generally more or less reddened, and it may become blue in cyanosis, or pale where anæmia exists. It must also be borne in mind, that the tongue is liable to be discoloured by particular kinds of food, such as coffee, or milk, and still more markedly by swallowing such medicines as the various preparations of iron.

Finally, the tongue is liable to be the seat of new formations—syphilitic fissures, ulcers, and deposits are frequently met with, and cancerous disease of that organ is not uncommon.

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\* Virch. Arch., vols. lxx. and lxxiii.

*The sense of taste* will be considered under the nervous system.

**Odour of the Breath** is often an important indication. Offensive breath may be the result of indigestion, but more commonly arises from some disease of the mouth, such as carious teeth, decomposing matter in the crypts of the tonsils, or ulceration of the gums. The peculiarly offensive odour caused by ozæna usually disappears from the breath when the nostrils are compressed, and the patient breathes through the mouth alone; of lung affections, those which most commonly cause offensive breath are gangrene and bronchiectasis.

**Saliva.**—The *reaction* of the mixed saliva found in the mouth is normally alkaline, but it becomes acid when retained for long in the buccal cavity, in dyspepsia, and in diabetes. It is mixed with epithelium and leucocytes, and often contains certain vegetable organisms (*Oidium*). A small quantity of albumen is present in health, and may be much increased in disease.\* Certain medicines when administered internally appear in the saliva. In particular, the salts of iodine and bromine; urea is also sometimes present in cases of uræmia, but bile and grape sugar are never excreted in the saliva. In disease the saliva may be increased or diminished in quantity.

*Increase of Saliva* (salivation or ptyalism) may arise—

1. From irritation in mouth and throat; as in stomatitis (mercurial or simple), gum-boil, ulcers, dentition, sore throat, &c.
2. From irritation in stomach, pancreas, intestines, uterus; as in cases of dyspepsia, worms, and in pregnancy.
3. From neuralgia, especially of the face.
4. From certain diseases of the brain, medulla, and spinal

\* In catarrh of the mucous membrane of the mouth, the buccal mucus contains large numbers of epithelial cells and leucocytes, and a great quantity of dissolved albumen.

cord. In insanity, hydrophobia, hysteria, and particularly in bulbar paralysis.

5. From the action of certain drugs, in particular, mercury and jaborandi.

*Diminution of Saliva* is chiefly met with in fevers and in diabetes. It occasionally results from blocking of the salivary duct with a calculus. It may also be diminished by mental emotions, and by the administration of certain drugs, such as atropin, daturin, and Jod-ethyl-strychnine.

**Fauces.**—The examination of the fauces may be conducted in two ways,—by inspection and by palpation.

*Inspection.*—The patient should be placed opposite the window, and made to open his mouth widely, while the physician stands in front of him and a little to one side. In most cases it is necessary to depress the tongue by means of a spatula or the handle of a large spoon, and this should be done in such a way that the instrument presses firmly on the horizontal part of the tongue without coming in contact with the soft palate. Sometimes a better view may be obtained by using a reflecting mirror (the laryngoscopic mirror answers admirably), in which case the patient must be placed with his back to the light.

*Palpation.*—It is best to stand at the patient's right side, and to introduce the fore-finger of the right hand into the mouth, pressing at the same time with the left hand on the soft parts behind the angle of the jaw. This bimanual method allows the physician to appreciate very accurately the condition of the parts in question.

By a careful use of these two methods we must satisfy ourselves of the condition of the pillars of the fauces, with the tonsils lying between them (in health barely visible); of the soft palate arching up on either side, with the uvula depending from the centre of the posterior wall of the pharynx behind and of the epiglottis. The mucous membrane of all these parts is moist, and has a red appearance, the colour being particularly deep over the soft palate. On the posterior wall of the pharynx,

the vascular ramifications are usually very distinct. In examining the fauces in this way, we have to note changes in the mucous membrane in regard to colour, moisture, and smoothness of surface. The presence of abnormal secretions, of ulcers, swellings, tumours, false membranes, and other pathological changes, will thus also become evident. Finally, a careful observation ought to be made of the movements of the soft palate, and this is best done by making the patient say "a." We must look for enlargement, inflammation, or ulceration in all these parts, and for changes in the mucous membrane covering them. Apart from mechanical and chemical causes, inflammatory redness of the fauces may occur in many general diseases. It is, however, perhaps most marked in scarlet fever, in which disease throat symptoms occur early. The redness usually beginning in the middle of the soft palate, quickly spreads over the whole mucous membrane of the fauces, and becomes very intense, being accompanied with considerable swelling of these parts. The other exanthemata are also frequently accompanied with sore throat, but the redness is less intense. In secondary syphilis, an erythematous reddening of the fauces is not uncommon, the eruption being often distributed symmetrically on either side of the soft palate. More chronic inflammatory conditions give rise to a dark-red appearance of the mucous membrane, in which the follicles may be more conspicuously affected; and care must be taken not to mistake the distended mouths of follicles for ulcers.

In acute tonsillitis the tonsil on the affected side becomes much swollen and of a dark-red colour, as does the corresponding side of the soft palate and the uvula, and these parts are usually found to be covered with a tenacious muco-purulent coating. Along with these appearances, the voice assumes a very characteristic thick nasal tone, the jaws are opened only with difficulty, and the act of swallowing is very painful. The formation of a tonsillar abscess is a common result of this process.

*Ulceration* of the throat is most commonly of the following

varieties:—(1) catarrhal; (2) follicular (corresponding to the follicles); (3) scarlatinal; (4) syphilitic; (5) diphtheritic; (6) herpetic; (7) cancerous; (8) result of the action of hot water, of acids, alkalis, or other chemical substances. The bleeding which sometimes occurs as the result of ulceration, must not be mistaken for hæmoptysis.

*Diphtheria* is, perhaps, the most important of all the affections which are met with in this neighbourhood. The tonsils are usually the first part attacked. They become red and swollen, and over the mucous membrane covering them there forms a false membrane, which is at first white, but which gradually assumes a dirty greyish colour. This membrane quickly spreads to the soft palate and uvula, and ultimately to the posterior wall of the pharynx, so that at last the whole fauces may be over-spread. When at any point this false membrane is forcibly detached, the mucous membrane below it will be found denuded of its epithelium, raw and bleeding. Over this area the false membrane quickly becomes renewed. The diphtheritic false membrane must be distinguished from certain other pathological conditions which more or less closely resemble it, such as patches of secretion exuded from the crypts of the tonsils, or layers of purulent secretion on the fauces in chronic catarrh. If there be any doubt, the microscope will at once show that these do not possess the characters of a fibrinous false membrane. The white patches of thrush may also be mistaken for diphtheria, but may be distinguished by showing under the microscope the presence of the oidium (see fig. 2, p. 28). The diagnosis of diphtheria is, however, always to a large extent founded upon the severe general symptoms which present themselves,—the swelling of the cervical glands, the great prostration without high fever, and the albuminuria. A slight or doubtful case is often shown to have been diphtheria by the subsequent occurrence of post-diphtheritic paralysis, which is usually found in connection with the soft palate (showing itself by the regurgitation of fluid into the nostrils during swallowing), but sometimes affects the limbs.



Tumours in the region of the fauces are usually more of a surgical than of a medical nature. Reference must, however, be made to nasal polypi which sometimes hang down into the throat, to tumours of the epiglottis which may show themselves above the root of the tongue, and to the various forms of malignant growths which may be met with in connection with the pharynx. These usually present little difficulty in the way of diagnosis. Bulging forward of the posterior wall of the pharynx may be produced by retro-pharyngeal abscess—usually the result of caries of the cervical vertebræ—similar prominence in this neighbourhood may, in rare instances, arise from carotid aneurism.

*Movements of the Soft Palate.*—The exact innervation of the muscles of the soft palate is still somewhat obscure. Clinically, however, it seems clear that paralysis either of the spinal accessory or of the facial nerve may involve the palate. When the palate is paralysed in cases of facial paralysis, it shows that the lesion is above the geniculate ganglion, as the large superficial petrosal nerve (which supplies the palate) branches off at that point. Occasionally the paralysis of the palate is of central origin, as in hemiplegia, but more frequently it is due to some affection of the nerves in their course, or is peripheral in its origin. Perhaps the most frequent cause is diphtheria. The appearance which the palate presents depends upon the degree of its involvement. When one lateral half of the palate is paralysed, it will be seen that the affected side is further forward, that the raphe is drawn backwards and towards the sound side, and that the arch of the palate has become non-symmetrical, the highest point lying more posterior and nearer the sound side. When the paralysis is bilateral, the palate is lax and immobile, and hangs far forward. Rarely, cases come under observation in which the paralysis is limited to one or two of the palatine muscles, as, for example, the azygos uvulæ. When that muscle is paralysed the uvula usually becomes bent round to the sound side.

Diphtheritic paralysis of the palate affords the best example of the symptoms which follow such paralysis. When the soft

palate is so far paralysed as to prevent the proper closure of the posterior nares, it is found that, during swallowing, the fluid passes into the nose and streams out through the nostrils. The speech also becomes nasal and indistinct. Anæsthesia of the palate often accompanies paralysis. Hyperæsthesia is very common as the result of chronic catarrh, abuse of alcohol or tobacco, and particularly in hysteria.

**Mastication** may be rendered difficult or painful by the presence of inflammatory affections of the lips, gums, cheeks, or tongue, by defective teeth, by cancerous or other ulceration of those parts, or by paralysis or spasm of the muscles employed in the act. The buccinator and the orbicularis oris receive their motor supply from the seventh nerve; and when that nerve is paralysed, the food accumulates between the cheek and the teeth. The motions of the tongue are affected in paralysis of the hypoglossal nerve. The muscles of mastication (masseters, temporals, pterygoids) are innervated by the motor branch of the fifth nerve. Their movement may be interfered with in two ways, either by spasm or by paralysis. *Masticatory spasm* may be tonic or clonic. The tonic form, one of the initial symptoms of tetanus, shows itself by the continuous and rigid contraction of the muscles of mastication, resulting in firm closure of the mouth, the lower jaw being drawn upwards in contact with the upper, and at the same time forced somewhat backwards. Clonic spasm results in the rapid rising and falling of the lower jaw, which produces a sort of chattering of the teeth similar to that seen in ague. Masticatory spasm may be due to such affections as chorea, epilepsy, and hysteria, besides tetanus, as already mentioned. It may also arise reflexly from some peripheral irritation of the fifth nerve. *Masticatory paralysis* shows itself in inability to chew, which is more or less pronounced according as the condition is bilateral or unilateral. It usually owes its origin to central causes, such as bulbar paralysis, and affections of the cortex cerebri.

**Deglutition** may be rendered difficult and painful by various

affections of the mouth and tongue, such as swelling, ulceration, inflammation, &c., but apart from such temporary causes, it may arise from paralysis of various groups of muscles. For purposes of description, the act of deglutition may be divided into three stages: (1.) The gathering up of the food into a bolus and thrusting it through the anterior pillars of the fauces. (2.) Its passage through the upper part of the pharynx until it has passed the orifice of the larynx. (3.) Its descent through the lower part of the pharynx and the œsophagus. The first stage of deglutition is interfered with in paralysis of the hypoglossal nerve, for the tongue cannot then form and force back the bolus into the fauces. Such paralysis is almost invariably of central origin. In the second stage of deglutition, disease of the facial nerve above the geniculate ganglion, by causing paralysis of the soft palate, allows food to pass into the posterior nares, and the same symptom is present in post diphtheritic paralysis, in syphilitic ulceration of the palate and epiglottis, and in cleft palate. This second stage of swallowing may also be interfered with by paralysis of the muscles of the pharynx. The food sticks at the root of the tongue, and occasions such dyspnœa as to require its removal by means of the finger, and fluids pass readily into the larynx. This paralysis, rare as a peripheral disease, is common as a result of affections of the pons and medulla, and of diseases of the base of the brain, compressing the cranial nerves. The third stage of deglutition may be interfered with by mechanical obstruction of the œsophagus (impacted foreign bodies, pressure of aneurism or other tumour, simple or cancerous stricture, &c.), or by paralysis of the muscles of the œsophagus, which is very rare as an isolated affection. In the latter case, solids may manage to make their way down the tube by their own weight. Spasm of the œsophageal muscles sometimes prevents deglutition in cases of hysteria.

*Examination of the œsophagus* is practically limited\* to mediate palpation by means of the œsophageal bougie. It is

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\* The œsophagoscopes of Störck and Mackenzie can hardly be said as yet to have proved of practical value in diagnosis.

most convenient for the purposes of diagnosis to use the elastic tube of a stomach pump. The tube must be softened in warm water, lubricated (preferably with glycerine or white of egg), and held in the right hand as one holds a pen, passed gently over the back of the patient's tongue, being guided by the left forefinger of the operator over the epiglottis, and then gently and steadily pushed into the stomach. Of the difficulties and dangers which the operator may meet in the course of this exploration, the following are the most important:—The œsophagus must never be sounded until there is certainty that no aneurismal tumour is pressing upon it, into which the point of the tube might be forced, nor in cases of gastric ulcer. In ordinary circumstances there is but little danger of the instrument passing through the glottis into the trachea, but whenever paralysis and anæsthesia of the laryngeal structures exists, very great care must be taken. It is hardly necessary to warn the student of the danger of forcing the sound through the œsophageal structures. Such an accident has happened not unfrequently, and it is of course more ready to occur when the walls are softened by cancerous deposit.\*

In sounding the œsophagus we may meet with—

1. *Pain*.—If the pain be felt again and again at the same spot, it indicates a local process, probably of an inflammatory nature. The presence of ulceration may be conjectured if the sound, however carefully it has been introduced, always comes away smeared with blood.

2. *Obstruction*.—This may be caused by the point of the sound passing into a diverticulum, and it is characteristic of this condition that the instrument sometimes passes with great ease, and sometimes is absolutely arrested. Strictures of various kinds also prevent the passage of the sound. The purely spasmodic strictures met with in hysteria may be distinguished from

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\* The use of the stomach tube to remove fluid from the stomach to be examined for purposes of diagnosis will be considered further. Since, in this case, a very soft rubber tube may be employed, its use is not subject to all the restrictions mentioned here.

those due to organic disease, by placing the hysterical patient under the influence of chloroform, when an instrument which before was obstinately resisted, now passes freely into the stomach.

*Auscultation of the Œsophagus* was first employed as a means of diagnosis by Hamburger.\* The stethoscope is to be applied a little to the left of the spinal column in the cervical or dorsal region, and the sound listened to which arises in the œsophagus when the patient swallows water. In health, the act of deglutition is accompanied with a short, clear, gurgling sound. When, however, œsophageal obstruction exists, this sound is prolonged, and altered in character below the seat of the stricture.

**Appetite.**—Derangements of the appetite may occur both in general diseases and in local affections of the stomach and intestines.

*Anorexia*, or loss of appetite, is present during all acute febrile diseases, and may also result from excessive fatigue of mind or body, or from depressing emotions, such as pain or grief, as well as from the use of narcotics or alcohol. It is also caused by inflammatory affections of the stomach, cancer of the gastric walls, constipation, and other abnormal conditions of the intestines.

*Boulimia*, or excessive appetite, may result simply from the habit of over-eating, or may be due to the presence of worms in the stomach or intestines. It is also present in certain chronic inflammatory conditions of the gastric mucous membrane, and is a prominent symptom in the course of diabetes, and in various nervous disorders (insanity, hydrocephalus, epilepsy, hysteria, and hypochondriasis).

*Pica*, or depraved appetite, in which the patient craves for various abnormal and even disgusting articles, is sometimes met with during pregnancy, in patients suffering from chlorosis and mania, and in idiots.

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\* "Klinik der Œsophagus Krankheiten," 1871.

**Thirst** almost invariably attends all feverish states of the system. It is a marked symptom in diabetes ; and in irritative conditions of the stomach thirst commonly appears some hours after eating.

**Sensations during Fasting.**—In the atonic form of chronic dyspepsia there is frequently before meals a feeling of sinking in the epigastrium, along with faintness. Pain, when the stomach is empty, is sometimes due to cancer or gastric ulcer, although in these diseases the greatest pain is usually felt after food has entered the stomach. Over-secretion of gastric juice may give rise to pain when the stomach is empty, which is then usually relieved by eating.

**Sensations after Eating.**—Painful sensations after food is swallowed, which are referred to the gastric region, vary from the slightest feeling of discomfort or oppression up to the most severe agony. The pain may be due to—

1. *The Presence in the Stomach of Irritating Substances.*—This irritation may be mechanical, when indigestible food has been swallowed ; or chemical, due to the presence of corrosive poisons, or, more commonly, of the various abnormal products which are formed in the stomach when digestion is imperfect. In its slighter forms, such irritation gives rise merely to a feeling of weight, discomfort, or distension ; in its graver varieties the pain may be very great.

2. *Organic Diseases of the Gastric Wall.*—Of these the most important are cancer and gastric ulcer. In both of these affections, especially in the latter, pain becomes very severe almost immediately after food has been swallowed, and continues for a considerable time, though, as a rule, the patient is free from pain when the stomach is empty. In gastric ulcer the pain is sometimes sharply localised, and is increased by external pressure. Extensive organic disease may, however, in rare cases, run its course without pain.

3. *Nervous Causes.*—Pain in the stomach is occasionally of

a purely neuralgic character, due sometimes to hyperæsthesia of the gastric mucous membrane (as in chlorosis), at other times to changes in the brain or spinal cord. The most marked example of the latter variety of gastralgia is to be found in the "gastric crisis" of locomotor ataxia.

The name *heartburn* has been given to a peculiar pain of a hot or scalding character, referred usually to the region of the epigastrium. It is due to abnormal digestion, and is frequently accompanied by acid eructations. There are various other forms of pain occurring in the region of the stomach, which may be mistaken for gastric pain. *Hepatic colic*, due to the passage of gall-stones, is very difficult to distinguish at its commencement, although, even then, bile pigment may be found in the urine. When the accompanying jaundice appears, a correct diagnosis will be easily arrived at. *Muscular rheumatism* may be distinguished by the long persistence of the pain, and by its rapid (though only temporary) removal on the application of the Faradic current. *Intercostal neuralgia* may be recognised by the hyperæsthetic condition of the skin which accompanies it. The pain of acute peritonitis, or of abdominal aneurism, will hardly be mistaken for pure gastric pain.

**Pyrosis**, or Waterbrash, is a common symptom of gastric catarrh, and consists in the sudden regurgitation into the pharynx of a mouthful of watery fluid, which is usually tasteless. This regurgitation is usually immediately preceded by sudden pain in the epigastrium. The pathology of this symptom is rather obscure. It would seem, however, most probable that the fluid comes from the pyloric end of the stomach.

**Flatulence and Eructation.**—The collection of gas in the stomach and bowels may result either from the swallowing of air, or from fermentative changes. It usually arises as a result of perversion of the digestive process in some of its various

actions. This gaseous distension of the stomach and intestines occurs most readily in cases in which the muscular walls of these viscera have become more or less paralysed—as in hysteria and the more severe forms of fever, particularly in typhoid and puerperal fevers. When it reaches a considerable degree, it is called tympanites or meteorism; and its seat, whether in the stomach or the bowel, may be determined by percussion, as will be afterwards explained. Meteoric distension often interferes with respiration, owing to the upward pressure which it exerts on the diaphragm. This acts in two ways, partly by compressing the lungs and limiting the respiratory movements, and partly by mechanically interfering with the free motion of the heart. This distension is usually relieved by the expulsion of the gas either by the mouth (eructation) or the anus. Eructation usually takes place suddenly and with some force, so that the gas, rushing up the œsophagus into the pharynx, often carries with it some of the liquid or solid contents of the stomach. Gaseous eructations have been analysed at various times, and have been found to contain carbonic acid gas, hydrogen, marsh gas, olefant gas, oxygen, and nitrogen.\*

**Colic** is a frequent accompaniment of flatulence. Its chief symptom is pain, which is usually situated near the umbilicus, but which diffuses itself in different directions. This pain, which is usually of a sharp, twisting character, comes in distinct paroxysms, which gradually commence, reach a maximum, and as gradually fall away again. This character of the pain, as well as the slowing of the pulse which accompanies it, and the normal temperature, suffice to distinguish colic from peritonitis, rheumatism, &c.

**Methods of directly testing the Digestive Power.**—Much more trustworthy for diagnostic purposes than these subjective sensations is the examination of a specimen of the contents of

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\* *Vide* analysis by Ewald and Rupstein, *Arch. f. Anat. u. Physiologie* 1874.



the stomach, removed for that purpose by means of a stomach tube. It is only recently that this method of diagnosis, which we owe to the labours of Kussmaul and of Leube,\* has come into use. For this purpose a very soft rubber tube must be used, so as to make the operation free from all danger. Leube recommends that this tube should have a diameter of 1 cm., and a bore of .55 cm. Such an instrument it is safe to pass into the stomach in almost all cases, save only where there is a gastric ulcer. It is, however, so soft and yielding that it requires to have a stiff sound pushed into its bore so as to allow it to be directed past the laryngeal opening. So soon as it has entered the œsophagus, this inner sound must be withdrawn, and the soft tube pushed onwards into the stomach. The general rules for introduction of the instrument have been already given (p. 37). The upper end of this stomach tube is connected by means of a T-tube with (1) a glass vessel containing water, and (2) an empty vessel for receiving the contents of the stomach. The branch tube leading to the reservoir is kept closed when not in use, by means of a clip. In this way we are furnished with a ready means of removing the contents of the stomach, and of washing out that viscus, if that be necessary. By means of this apparatus two important lines of inquiry may, in each individual case, be followed up: (1) the measurement of the duration of the digestive act, and (2) the estimation of the digestive strength of the gastric fluid.

1. *Method of determining the Duration of Digestion.*—Leube† has found that a healthy stomach will completely digest an ordinary meal in seven hours. He gives the patient whose digestion he wishes to test, a meal, consisting of a moderate quantity of soup, beef-steak, and white bread, and for the next seven hours allows nothing to be swallowed. At the end of that time the stomach tube is introduced, and the contents of

\* See, in particular, a masterly article by the latter in the *Deutsches Archiv. für klin. Med.*, vol. xxxiii. (1883).

† *Loc. cit.*

the stomach washed out. If the power of digestion be good, the fluid should be either clear, or at most slightly clouded with a little mucous. When, on the other hand, the digestion is bad, the stomach will be found to contain much of the undigested food. In this way it is often possible to make a diagnosis of nervous dyspepsia when along with many dyspeptic symptoms we find the rapidity of digestion normal.

2. *The determination of the Digestive Power of the Gastric Juice.*—In order to obtain a pure specimen of gastric juice for this purpose, the best method of procedure is as follows:—The stomach, when completely empty, is to be washed out with about 400 c.cm. of lukewarm water, until the returning stream is clear and neutral. After this operation is ended, 100 c.cm. of iced water are then passed into the stomach, and allowed to remain there for ten minutes, after which the stomach is to be completely washed out with 300 c.cm. of ordinary water. The cold stimulates the secretion of gastric juice, and this is removed in the last washing. It is now necessary to test the gastric juice so obtained in two ways: (a) in regard to pepsin, and (b) in regard to acidity.

(a.) *Pepsin.*—Of this fluid 30 c.cm. are placed in a test-tube, to which is to be added a small mass of coagulated albumen.\* The test-tube is then to be introduced into an incubator, and if the gastric juice in the particular instance contains a proper amount of pepsin, the albumen will be rapidly dissolved. It is necessary to acidulate the fluid if its reaction is not already acid.

(b.) *Acidity.*—The acidity of the fluid is to be tested by means of tincture of litmus in the usual way. In health it will be found to react distinctly acid.

The importance of these inquiries in regard not merely to diagnosis, but also to treatment, will be readily perceived. For fuller details the reader may be referred to the article by

\* It is best to cut the boiled white of an egg by means of a double parallel-bladed knife, and then with a cork-borer to punch small discs out of these slices.

Leube already noted, and to various contributions by Von den Velden.\*

**Nausea and Vomiting.**—The expulsion of the contents of the stomach in vomiting is caused by the forcible contraction of the abdominal muscles, diaphragm, &c., the gastric muscular fibre itself probably remaining inactive. It is a reflex act, having its centre in the spinal cord. This reflex centre corresponds apparently to a considerable portion of the cord, and includes part of the respiratory centre. Vomiting may be produced in various ways: (1) By any disease of the brain or spinal cord, which involves this reflex centre; (2) by excessive irritation of the respiratory centre, in violent coughing, &c.; (3) by the presence in the blood of any substance which may occasion irritation of the centre or of the terminal twigs of the vagus, such as effete products in uræmia, emetics, &c.; (4) by irritation of the vagus or its branches. This is by far the most frequent cause of vomiting. It is thus that vomiting is to be explained in cases of uterine disease or pregnancy, kidney disease, the passage of gall-stones, tickling of the soft palate, and, above all, in affections of the stomach itself. Chemical or mechanical irritation of the gastric mucous membrane gives rise to vomiting. It may also be produced by fermentative changes in the contents of the stomach, by ulceration, or chronic catarrh of the gastric mucous membrane. Gastric vomiting is usually preceded by nausea and pain, and bears some relation to the food swallowed; whereas, when this symptom arises from irritation of other kinds, these are frequently absent. The diagnostic significance of vomiting can, however, be best appreciated after the examination of the

**Vomited Matter.**—This consists of the contents of the stomach and sometimes of the duodenum. The fluids and solids vomited are more or less acted upon by the gastric juice, and are usually strongly acid. Of abnormal substances present

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\* *Deutsches Archiv*, vols. 23 and 26; and *Berl. kl. Woch*, 1877.

the most important is *blood*, which may be almost pure, but is generally more or less acted upon by the gastric juice, and thereby coagulated and darkened so as to resemble the grounds of coffee, the hæmoglobin being broken up into globulin and hæmatin. Hæmatemesis (the vomiting of blood) is most frequently met with in connection with gastric ulcer or carcinoma; but it also occurs in blood diseases (yellow fever), in congestion of the veins of the stomach (cirrhosis of the liver, pressure on inferior vena cava), and sometimes vicariously, when the menstrual flow is arrested. Finally, it may result from wounds of the stomach, or from the bursting of an aneurism into the stomach or œsophagus. There are mainly two conditions which are apt to be mistaken for hæmatemesis—viz., bleeding from the nose, and bleeding from the lungs. The former is only difficult of diagnosis in cases where the blood has been first swallowed and then vomited again, but an examination of the nose and throat will almost always make plain the source of the hæmorrhage. Bleeding from the lungs is more difficult to distinguish from hæmatemesis. The main points of distinction will be enumerated under the head of "Sputum." Bile is frequently found in the vomited matter, rarely pus, and still more rarely faecal matter. The latter usually points to intestinal obstruction, although, according to Bamberger, it may occur independently of such a condition when the bowel is paralysed as a consequence of peritonitis or typhoid fever.

*Microscopic examination* of the vomited matter shows the various substances derived from the food swallowed, sometimes *Sarcinae ventriculi* and intestinal parasitic worms (*Ascaris lumbricoides*, &c.). The *sarcinae* are small cube-shaped cells united in groups of four. They are met with in nearly all cases in which food remains too long in the stomach and there ferments, particularly where



FIG. 3.—*Sarcinae*. (Roberts.)

the stomach is dilated. Other micro-organisms are also found, but, so far as our present knowledge goes, they are of little diagnostic importance.

**Defæcation.**—While normally defæcation occurs with regularity once in twenty-four hours, it is not uncommon to find persons who have two motions in that period, or whose bowels act only once in two days, without the bounds of health being over-stepped. In infants, the bowels move frequently—four or five times a-day. The following points have to be inquired into :—

1. *The Frequency of the Motions*, and the period at which the bowels act relatively to eating, drinking, exercise, &c.

2. *The Character of the Act of Defæcation.*—Faintness or sickness may precede the act, which may itself be painful and straining, and may be followed by a sensation of the rectum not having been emptied of its contents (tenesmus). The actual condition of the anus and rectum must be determined, and the presence of piles, fissure, prolapse, ulceration, &c., looked for.

The two conditions of constipation and of diarrhœa demand brief notice.

*Constipation* may result from—

1. Mechanical obstruction ; and this may be caused in various ways, such as from accumulations of various kinds in the bowel, by cicatricial or cancerous stricture, by external compression of the intestines, by strangulation or intussusception, and by spasm or paralysis.

2. Defective peristaltic action.

3. Deficiency of the secretions.

These two last causes may arise from too frequent use of purgatives, from neglect of the regular performance of the act of defæcation, from the abuse of opium, from sedentary and from enervating habits, as well as from derangement of stomach and liver, and from many other causes too numerous to mention.

*Diarrhœa.*—The causes of diarrhœa may be grouped as follows :—

1. While the intestinal canal is normal, diarrhœa may be excited by abnormally strong stimuli, such as improper food, hardened fæces, purgatives, or the presence in the bowels of noxious materials in course of elimination from the body in such diseases as uræmia and gout.

2. There may be abnormal irritability of the intestinal nervous system to such an extent that the normal stimulus produces so much peristaltic action as to lead to diarrhœa. This condition is found in connection with nervous disease, and in individuals of a nervous temperament. It further occurs when the nerves of the bowel are laid bare by ulcerative processes, and are thus more easily irritated.

3. Pathological conditions of the mucous membrane may lead to the pouring out of much secretion into the bowels. This group includes all the grave affections of the intestinal tract, and also many general diseases.

### Character of the Fæces.

#### 1. *Macroscopic Characters.*

(a.) *Colour.*—The normal colour of the fæces may be altered by reason of the food eaten: becoming light in colour with milk diet, and dark brown when much meat is taken. Still more marked is the influence of various medicinal agents upon the colour. Iron and bismuth, when taken internally, blacken the motions, while the administration of preparations of iodine causes a blue, calomel a green, and logwood a red-brown colour to appear in the fæces. If all these causes of altered colour be excluded, then the alteration is dependent upon bile or upon blood. The presence of bile gives rise to a yellow or to a green tint, and in its absence the fæces assume a grey or chalky appearance.\* Where there is profuse diarrhœa the evacuations become very pale in colour, owing to the dilution of the bile. The “rice

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\* This chalky appearance is not merely to be traced to the absence of bile pigment, but is likewise due to an abnormal quantity of fat, the result of the disturbance of the digestion of fat, in which process bile plays an important part.

in large amount, when not accounted for by diet, usually indicates affection of liver or pancreas. To the presence of blood allusion has already been made.

*Microscopic Examination of the Fæces.*—Fragments of food, including muscular fibre, connective tissue, fat cells and crystals, coagulated albumen, vegetable cells, &c., are readily to be detected in the stools. Besides these, we have to recognise certain elements which are derived from the tissues themselves, as for example, epithelial cells, mucous and pus corpuscles, blood

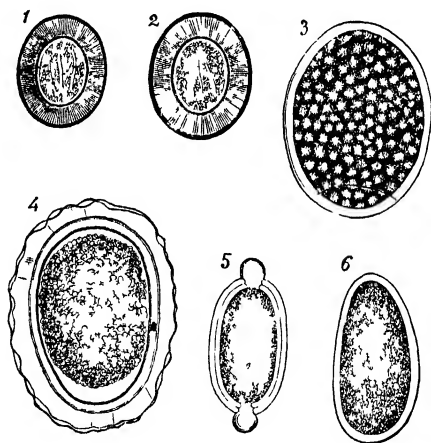


FIG. 7 — EGGS OF THE FOLLOWING FORMS —

- |                                 |                                |
|---------------------------------|--------------------------------|
| 1. <i>Tænia solium</i> .        | 4 <i>Ascaris lumbricoides</i>  |
| 2. <i>Tænia mediocanellata</i>  | 5 <i>Trichocephalus dispar</i> |
| 3. <i>Bothriocephalus latus</i> | 6 <i>Oxyuris vermicularis</i>  |

corpuscles and crystals of various forms—triple-phosphate, cholesterin, charcot's crystals, hæmic crystals, and balls of leucin. In the stools there also may be found many vegetable and animal parasites, the former including *Bacterium termo*, *Sarcina ventriculi*, and similar forms, while to the latter belong *Amæba coli*, various infusoria, and the eggs of the parasitic worms, the adult forms of which have been already described.

The general form of the more important of these is illustrated in the accompanying woodcut (Fig. 7).

In how far, from an examination of the fæces, the physician may infer what is the exact condition of the intestinal tract, is a question of some difficulty. The following points, which may be of use in localising catarrhal affections of the intestines, are, in great measure, derived from an article by Professor Nothnagel ("Zeitschrift für Klinische Medicin," vol. iv., 1882).

The evacuation of pure mucus from the bowel without any admixture of fæces points to catarrh of the rectum. When firm fæces are passed completely enveloped in mucus, we may conclude that the morbid process affects the lower part of the colon and the rectum. The admixture of mucus with the fæces in abnormal quantity is not always apparent to the naked eye. It often happens, that when the fæculent matter is examined microscopically there are found scattered intimately through it small masses of mucus, which are whitish-grey, hyaline, and transparent. This peculiar admixture of mucus indicates that the catarrhal affection is limited to the upper portion of the large intestine (and, possibly, the small intestine), while the rectum and descending colon are free from disease. When the stools contain small masses of mucus tinged yellow with bile pigment, we may conclude that the small intestine has become affected.

The "rice-water" stools of *Asiatic Cholera* are so characteristic as to possess very considerable diagnostic value. The name is derived from the fact that small lumps of epithelial cells are suspended in a more or less clear fluid, giving it something of the appearance of rice-water. There is little or no fæcal odour. In *cholera nostras*, however, stools closely resembling these are occasionally met with.

It has been asserted that in the stools of true Asiatic cholera the characteristic curved bacillus or spirillum, now so well known as Koch's "comma bacillus," can always be found by appropriate methods. In some cases this is so, but there is no



doubt that it is not so constantly present as to allow of its possessing the diagnostic importance ascribed to it by Koch. In many cases of absolutely characteristic Asiatic cholera the most careful examination of the intestinal contents and of the stools fails to give evidence of the presence of this micro-organism.

In cholera nostras a micro-organism resembling in some respects the comma bacillus of Koch has been found, but it is clearly a different variety.

## CHAPTER III.

### Examination of the Abdomen.

#### INSPECTION.

THE physical examination of the abdomen can only be made with advantage when the patient is in the recumbent posture. It is well to have the shoulders slightly elevated by means of a pillow, so as to relax the abdominal muscles, which can be still further effected by causing the knees to be raised. The examination is conducted by means of inspection, palpation, percussion, and auscultation, each of which will be considered in turn.

The abdominal surface has been arbitrarily divided into certain regions, the position of which is sufficiently indicated in the accompanying diagram.

The form of the abdomen varies greatly within physiological limits. A full dietary and a corpulent habit cause prominence in this region, whilst in old age and after prolonged inanition, the belly sinks in, and its bony walls become unduly prominent.

The pathological changes which inspection indicates may be

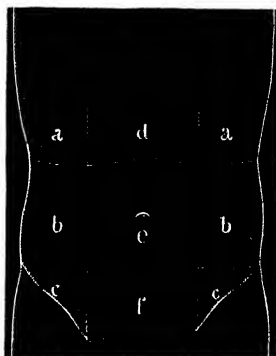


FIG. 8.—REGIONS OF THE ABDOMEN.

- (a.) Hypochondriac regions.
- (b.) Lumbar regions.
- (c.) Iliac regions.
- (d.) Epigastric region.
- (e.) Umbilical region.
- (f.) Hypogastric region.

considered under three heads—(1) General prominence; (2) General retraction; (3) Local tumefaction.

*General prominence* of the abdomen is found—

1. In *ascites*, where an effusion of fluid has taken place into the peritoneal sac. The fluid gravitates to the most dependent position; and, consequently, when the patient lies on his back, the anterior part of the abdomen is flattened while the sides bulge, whereas, if the erect position be assumed, the prominence is greatest in the hypogastric region.

2. In *meteorism*, or-accumulation of gas in the intestines. In this case a change in position does not affect the form of the abdomen, which is more spherical than in the former condition.

When the abdomen is greatly distended, from whatever cause, the diaphragm becomes raised, the ribs pressed outwards, and the position and character of the apex-beat of the heart altered. The abdominal walls become smooth and glistening, the recti muscles are pushed asunder, and in the interval between them the peristaltic motion of the intestines may occasionally be observed. The umbilicus rises, first of all, to a level with the adjoining skin, and subsequently protrudes beyond it. The pressure exerted on the inferior vena cava gives rise to the development of the collateral venous circulation as a delicate blue network over the abdominal parietes, whilst obstruction to the portal circulation occasions distension of the veins at the umbilicus—this varicose condition being the more observable by reason of the prominence of the navel.

*Retraction* of the abdominal walls is met with in cases of inanition from whatever cause (particularly in œsophageal obstruction), and in all wasting diseases. It is also seen in various diseases affecting the nerve centres (as, for example, in tubercular meningitis), and is then attributable to contraction of the intestines, determined by the irritation at the base of the brain.

In such cases the bony walls become prominent; the vertebral column, with the pulsations of the aorta lying on its left side, may be seen, and the relaxed abdominal walls form pendu-

lous folds, between which friction may produce ulceration of the skin.

*Local tumefaction* may occur in connection with various abdominal organs as follows:—

*Stomach.*—Dilatation of this viscus gives rise to an oval swelling occupying the epigastrium, and extending chiefly towards the left. The position of the greater curvature may be indicated by a diagonal furrow running from the right downwards, and to the left.

The outline of the stomach is best appreciated when it is artificially distended, according to the method originally recommended by Frerichs. The patient is made to swallow successively a solution of tartaric acid, and one of bicarbonate of soda, of each salt as much as may be carried on the point of a table-knife. The result of the mixture of these solutions in the stomach is the development in that viscus of a large quantity of carbonic acid gas, and consequent distension.\* In a few seconds the position of the greater curvature, and the general outline of the stomach, can be made out with great distinctness. When the small intestine and colon become rapidly filled with the liberated gas, we may with safety diagnose incompetence of the pylorus, resulting from ulceration or carcinoma, or perhaps occasionally from abnormal innervation.

Tumours of the stomach sometimes cause visible prominence of the abdominal wall. In some cases such tumours do not alter their position with the respiratory movements, and may thus be distinguished from tumours of the liver and spleen, which rise and fall with the respiration; but sometimes this is not so, the gastric tumour mass having contracted such adhesions to neighbouring parts that it moves synchronously with the diaphragm.

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\* This method is not dangerous to the patient, provided that cases of gastric ulcer be excluded. The worst symptoms which an overdose might occasion would be vomiting, dyspnœa, and slight cyanosis—all disappearing in a few moments.

**Liver.**—In the healthy adult\* the liver gives rise to no visible prominence of the abdominal walls. When enlargement takes place, it usually shows itself first under the margin of the ribs, in the right hypochondrium, unless the left lobe be chiefly affected, when the tumefaction appears in the epigastrium. The edge of the liver, or the tumour arising from that organ when visible, may be seen to rise and fall with the respiratory movements—an important diagnostic point.

**Splenic tumours**, when of large size, cause visible tumefaction of the abdomen generally, particularly on the left side.

Tumours of the *kidneys and omentum* do not, as a rule, cause visible swelling.

Ovarian tumours are usually at first lateral in position, but subsequently they may develop to so great a size as to distend the whole abdomen.

**Abdominal movements** other than those of normal respiration may be seen—(a) respiratory, (b) pulsatory, (c) peristaltic.

The *respiratory movements* affect the position of all tumours which are attached to the liver or diaphragm. Splenic tumours are also similarly influenced, but to a less degree. Tumours of the stomach and pancreas, &c., are not so affected, unless adhesions have been formed.

**Pulsations** of various kinds are met with in the abdomen, but their nature will be more conveniently considered in connection with the circulatory system.

**Peristaltic motions** of the intestines may be observed in persons in whom the abdominal walls are abnormally thin (as in ascites, *vide antea*), or where such vermicular movements are usually energetic, as in cases of intestinal obstruction.

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\* In children the liver is, normally, large in proportion to the size of other viscera, and occasions a considerable degree of fulness of the abdomen, extending from the lower border of the ribs on the right side to the level of the umbilicus.

## CHAPTER IV.

### Palpation of the Abdomen.

At no portion of the body is the skilful application of the hand of more essential service to diagnosis than over the abdomen. By carefully pressing with the hand (which should be warmed) at various points with a kind of gentle kneading motion, we obtain, by the sense of touch, information regarding—1st, the condition of the abdominal wall; 2nd, the size, form, consistence, and mobility of certain of the abdominal organs, and whether any tumour be present within the cavity of the abdomen; and 3rd, if there be general tumefaction of the abdomen, whether such distension is the result of accumulation of gas in the intestines (tympanites), or is due to the presence of serous or inflammatory exudation; and, in the latter case, whether such exudation be in the peritoneal cavity or be inclosed in a cyst of some kind or another.

The position of the patient is of the greatest importance. He must lie on his back, with head and neck slightly raised, and with the knees flexed and drawn up towards the abdomen. In most cases it is well to engage the patient in conversation while palpating the abdomen, as otherwise the abdominal muscles are usually involuntarily contracted, and the glottis closed. The air in lungs, retained there by the closure of the glottis, supplies the necessary resistance to this contraction of the abdominal muscles, and if this resistance be removed (as is best done by forcing the patient to open his glottis in speaking)

the muscles have nothing to contract upon, and consequently become flaccid. The relaxation of these muscles may be still further aided by diverting the patient's attention. Should there be necessity for it, the exhibition of chloroform will allow of a very perfect exploration of the abdominal cavity.

**Abdominal Walls.**—The temperature of the skin, the amount of subcutaneous fat, the presence or absence of cedematous or emphysematous swelling of the subcutaneous cellular tissue (see chap. i.) are readily recognised by the palpating hand, and require no special mention here. Localised swellings of the abdominal wall, due to the presence of tumours, of inflammation, or of abscess, may be mistaken for more serious affection of the abdominal organs themselves. The immobility of such swellings, their position being unaltered by the respiratory movements, or by a change in the posture of the patient, will generally suffice to distinguish them. In reality, the physician has seldom any difficulty in satisfying himself of the seat of the swelling, whether in the parieties or in the cavity of the abdomen. In difficult cases, such, for example, as when a deep-seated abscess over the liver simulates a hepatic abscess opening outwards, the history of the case, and the other signs and symptoms, suffice as a general rule to indicate the real seat of the abscess.

The abdominal muscles, and more especially the “recti,” present, when contracted, certain inequalities in thickness which are occasionally mistaken by the inexperienced for abdominal tumours.

The various hernial protrusions which are found in the umbilical, femoral, and inguinal regions belong more especially to the domain of surgery.

**Peritoneal Cavity.**—Acute general peritonitis gives rise to great pain and tenderness on pressure over the whole surface of the abdomen. The patient then usually lies on the back, with knees drawn up, partly in order to relax the abdominal

muscles, and partly to diminish the pressure of the bed-clothes. In chronic peritonitis a characteristic doughy resistance is usually to be felt over the affected part, accompanied with some tenderness on pressure. Transudation of fluid into the peritoneal cavity (ascites) gives rise to a feeling of fluctuation. This is best appreciated by placing the hand on one side of the abdomen, and giving a smart tap on the surface at a point diametrically opposite. The impulse of the wave so formed can usually be clearly felt when it reaches the opposite wall. If, however, the amount of fluid be small, no such undulation will be observed in the ordinary position. The patient may then be placed on his elbows and knees, when the fluid will gravitate to the anterior part of the sac, and fluctuation can there be obtained.

Friction vibration can also occasionally be felt between two roughened peritoneal surfaces. It may be synchronous with the respiratory movements, and this most frequently if the visceral and parietal layers over the liver and spleen be the seat of the roughness (particularly in carcinoma of the liver). Friction vibration can also be induced in such cases by moving the peritoneal surfaces against one another, and pressure will at all times increase the strength of the friction.

**Liver.**—In the healthy adult, as a rule, only the left lobe of the liver can be felt by the palpating hand, giving rise to a slight feeling of resistance in the epigastric region. On very deep inspiration, however, the edge of the right lobe may sometimes be made to project so far beyond the costal margin as to offer appreciable resistance to the fingers. In children, the liver is of such size as to be readily examined by palpation.

Either as a result of enlargement or of lowered position (due, for example, to the downward pressure of a pleural effusion), the liver may come within reach of the palpating hand, and then we have to examine the condition of its surface, the consistence of the organ, its size, and general shape.

The *surface* of the liver may be smooth or rough. In



amyloid and fatty degeneration, and in congestion, the surface of the swollen organ is smooth, a condition which is very readily recognised by palpation. In the case of cirrhosis, the uneven granular surface gives rise to a characteristic feeling of roughness when the abdominal wall is made to glide backwards and forwards over the surface of the liver. More marked irregularities of surface are found in carcinoma, the distinct nodules of which can be felt, and occasionally the umbilications which these nodules present.

*Tenderness* on pressure is met with in congestion and in all inflammatory affections of the liver, such as hepatic abscess, cirrhosis, catarrh of the bile ducts. In carcinoma it is often a very marked feature, although even in this affection it may be absent. There is usually no tenderness in the case of the waxy and the fatty liver.

The *Consistence* of the liver is somewhat increased in fatty degeneration, still more so in congestion, and to a very marked degree in waxy disease, when the lower edge may assume an almost knife-like sharpness. The presence of fluctuation will usually suffice to distinguish a hydatid tumour or an abscess from a solid growth.\*

The *size* of the liver varies greatly. In some cases, as in acute yellow atrophy, the organ recedes so far into the concavity of the diaphragm as to be out of reach of palpation. In other instances (congestion, waxy degeneration, &c.), the lower edge may be found as low as the symphysis pubis. It must be carefully borne in mind that the position of the lower border is no safe guide to the size of the liver unless it be taken along with the position of the upper margin as ascertained by percussion.

*Abnormalities in shape.*—The practice of tight-lacing not

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\* When a hydatid tumour lying near the surface of the liver is percussed, a peculiar and very characteristic tremor (hydatid fremitus) may be felt over it, due to the reflection, from side to side of the sac, of the undulations into which the fluid has been thrown. The use of the aspirator for the diagnosis of such tumours will be subsequently discussed.

only forces the liver downwards, but also frequently so compresses the hepatic substance as to give rise to a deep furrow marking off the lower portion of the right lobe. This furrow can be readily detected by palpation. Still more obviously is the shape altered by the presence of a large tumour, cancerous or hydatid, growing from some particular part of the organ. It is important to remember that hepatic tumours rise and fall with the respiratory movements, which is not the case with growths in the stomach, omentum, pancreas, colon, or kidney, unless they have become adherent to the liver or diaphragm.

Occasionally the gall-bladder may be felt as a small pear-shaped tumour projecting from beneath the lower edge of the liver. Pressure, by emptying it of bile, may cause it to disappear; and in rare cases the presence of gall-stones in the bladder may be ascertained by palpation.

**Spleen.**—In the normal condition the spleen cannot be felt, and this is due partly to its deep-seated position, and partly to the fact that the splenic tissue is too soft to offer resistance to the palpating fingers. When, however, it becomes so enlarged as to reach the extremity of the eleventh rib, or to pass beyond it, then the spleen can be readily recognised. Increase in size of the spleen takes place in numerous diseases, such as leucocythæmia, amyloid disease, recent syphilis, intermittent fever, typhus, enteric, and scarlet fevers, &c.; and in addition, all diseases which produce obstruction to the portal circulation, directly or indirectly (such as cirrhosis of the liver or heart disease), cause splenic congestion, and consequently, enlargement of that organ.

When the spleen is but slightly enlarged, it can be best felt by tilting it upwards from the lumbar region on to the palpating hand. A feeling of increased resistance may thus be appreciated, although the limits of the organ may not be felt with any distinctness.

As the organ increases in size it projects from beneath the margins of the ribs towards the umbilicus, and rises and falls

with the respiratory movements.\* The enlargement is proportionately the same in all diameters, and so the spleen retains its original shape. The splenic notch is readily felt, and is important as a certain indication that the tumour with which we have to deal is splenic. In leucocythæmia, the spleen may attain an enormous size, and fill up the greater part of the abdominal cavity. Except in very rare cases (hydatid disease and carcinoma of the spleen) the surface of the swollen organ is smooth, and there is seldom any tenderness on pressure.

The consistency of the spleen is greatly increased in amyloid disease and in leucocythæmia. In congestive enlargement it is not so resistant, and in acute diseases the tumefied gland is of a very soft consistence. During the exacerbations of intermittent fever the spleen undergoes perceptible enlargement, while in cases of splenic congestion from portal obstruction, loss of blood from the stomach or intestines causes diminution in its size.

**Pancreas.**—Tumours of the head of the *pancreas* are rarely met with, and are difficult of diagnosis, owing to the way in which the gland is covered by the coils of the intestines, and to some extent by the lower edge of the liver. Hardness in such cases can usually be felt to the right of the middle line above the level of the umbilicus. It is deeply seated, not freely movable, and unaffected by the respiratory movements. The disease (which is almost invariably carcinoma) is seldom limited to the pancreas, but attacks the retro-peritoneal lymphatic glands, and other neighbouring parts.

**Stomach and Intestines.**—Tumours of the stomach (usually carcinomatous) are most common at the pylorus. They are then readily felt as irregular nodulated masses in the umbilical region, tender to pressure, freely movable for the most part, and

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\* It is well to remember that splenic tumours alter in position with the movements of the body. This often helps in the diagnosis of such tumours from tumours of the kidney.

but little affected in position by the rise and fall of the diaphragm.\* When the greater curvature is the part affected, the tumour mass is found somewhat lower down and to the left. Tumours of the lesser curvature and cardiac end of the stomach are rarely felt during life, as they lie deeply in the concavity of the diaphragm.

When the stomach is dilated palpation usually gives rise to well-marked gurgling sounds, the *timbre* of which is characteristic.

Pressure over the stomach occasions pain in many diseased conditions of that viscus. It is most circumscribed in cases of gastric ulcer, and is often of great severity.

In the intestines the retention of fæces, chiefly in the large intestine, may give rise to localised swelling at various points. These nodular masses are of a doughy consistency, and to a large extent disappear after purgation. Catarrhal and inflammatory conditions of the colon are apt to give rise to inflammation in the neighbouring tissues, resulting in a swelling which is usually ill-defined, doughy, hard, and very tender on pressure. Such inflammatory processes usually take place round the cæcum (perityphlitis). Cancerous masses may occasionally be felt at various parts of the colon; the cæcum and sigmoid flexure being most commonly the seat of the disease. Peristaltic movements of the intestines are occasionally to be felt when the abdominal walls are thin or the movements very energetic, as in stenosis of the bowel.

**Omentum.**—Tumours of the omentum are rare. They are of very various nature: cancerous, tubercular, hydatid, &c., and when developed are readily felt through the abdominal wall. When affected with carcinomatous disease the omentum becomes thickened and retracted, and its lower hardened edge may occasionally be felt crossing the abdominal cavity.

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\* This last point is often deceptive, for if the tumour have contracted adhesions, it may move with respiration, as has already been said.

The **Mesenteric Glands** are frequently the seat of tumours. They may be simply enlarged, along with other similar glands throughout the body, or they may be affected with cancerous, tubercular or other deposits. They are smooth, hard, moveable tumours of regular form. Occasionally they become fused together, along with other neighbouring structures (loops of small intestines, retro-peritoneal glands, &c.), into masses of considerable size, which, overlying the aorta, may have imparted to them a pulsatile movement.

The **Kidneys** are not, in their normal condition, within the range of palpation; but when they leave their position, or when they increase greatly in size, they may be felt. A floating kidney—that is, one the attachments of which are so loose as to allow of its moving more or less freely through the abdominal cavity—is recognised by its possessing the size and the characteristic renal shape, as well as by its great mobility, and by the presence of the pulsating renal artery, which may sometimes be felt entering at the hilus. The diagnosis is rendered more certain by the aid of percussion, as will be hereafter noticed.

Enlargement of the kidney occurs in hydronephrosis, echinococcus, carcinoma, &c., and the tumour is smooth or nodular according to its nature. It is recognised by its position and relations, its immobility, and its cylindrical shape from above downwards. Inflammatory thickening round the kidney and perinephritic abscess may also be recognised by palpation.

The *urinary bladder*, when distended, forms a pyriform tumour above the pubes, and may reach the level of the umbilicus, or even, in extreme cases, to a still higher point.

*Ovarian tumours* are in most cases cystic, and may be so large as to distend the whole abdomen. When small in size they usually lie on one side only, and gradually cross the middle line to assume an apparently central position. Fluctuation is generally easily made out. The characteristics of such growths will be considered under the head of percussion.

*Uterine tumours*, physiological as well as pathological, may be felt above the pubes. Their consideration belongs to the domain of the gynecologist and obstetrician.

*Aneurism of the abdominal aorta* may affect that vessel in any part of its course. If it lie very deep in the concavity of the diaphragm, it may not be capable of being felt with any distinctness, but where it can be reached a pulsating tumour is readily recognised. The pulsation must be distinguished from that produced by a tumour lying on the vessel, which is to be done by noting its true expansile character when compressed laterally between the fingers. Aneurisms of the main branches of the abdominal aorta also occasionally occur.

**Aspiration.**—The use of the aspirator has often an important diagnostic value in connection with diseases of the abdomen. This is particularly the case with reference to abscesses (hepatic, perinephritic, &c.), hydatid cysts, and ovarian tumours. Experience has shown that the introduction of an aspirator needle into the abdomen (even piercing the liver or bowel) is quite safe, provided that it be of small size and properly cleansed, and that after having been introduced its course be not altered. In many cases an ordinary hypodermic syringe is all that is needful for diagnosis, but occasionally it may be necessary to pass more deeply than that instrument allows of, and then Dieulafoy's aspirator may be employed. Before introducing the needle, the apparatus should be washed out with carbolic solution, and the needle itself dipped in carbolic oil. The needle is then to be passed through the skin, and whenever the opening at the point is covered, a vacuum ought to be made in the needle, either by opening the stop-cock communicating with the exhausted syringe or receiver, in the case of the aspirator, or, if the hypodermic syringe be employed, then by simply drawing back the piston. This "previous vacuum" is strongly insisted upon by Dieulafoy, because it at once shows when the point of the needle enters fluid by the rising of that fluid in the syringe. If the operator waits before opening the

stop-cock or drawing back the piston until he thinks the needle has penetrated deeply enough, he may pass a needle through a layer of fluid without detecting it. This is impossible if the "previous vacuum" be present the needle.

The operator should hold the needle very steadily, and must push it onwards in one direction only. It is not permissible to alter its course after it has penetrated some distance. Should this become necessary the needle must be removed and re-introduced.

The condition of the fluid obtained should be ascertained by examination with the naked eye, and by means of the microscope. Pus is readily recognisable, and the hooklets of hydatids are easily distinguished microscopically. The distinctive characters of ovarian fluid, as distinguished from ascitic, will be mentioned hereafter.

## CHAPTER V.

### Percussion of the Abdomen.

THE theory of percussion will be considered hereafter, and need not detain us at this point. Its use in connection with the abdomen is to define the outline of organs which it is not possible to reach by means of palpation. As a rule it is best to use the forefinger of the left hand as a pleximeter, laying it upon the surface of the abdomen, and eliciting a note by striking with one or more fingers of the right hand. The change of note as one passes off such a solid organ as the liver on to air-containing viscera, is usually sufficiently obvious.

Percussion gives information concerning—

1. The Condition of the Peritoneal Sac.
2. The Outline of the Liver.
3. The Outline of the Spleen.
4. The Outline of the Kidneys.
5. The Condition of the Stomach and Intestines.

**Condition of the Peritoneal Sac.**—When transudation of fluid takes place into the peritoneal cavity, it does not, in the first instance, affect the percussion note over the surface of the abdomen, since the small quantity of serum which at first collects, gravitates towards the lowest portion of the sac; and whether this point lie in the pelvis or towards the spinal column (determined by the position of the patient, erect or supine), the collection is too far removed from the surfaces ordinarily subjected to percussion to allow of its influencing the note obtained.



As the quantity increases it gradually makes its presence manifest, causing a dull note to be heard on percussion over the lower parts of the peritoneal cavity. With further increase in the quantity of fluid, the dulness extends its area, until in extreme cases, where the sac is greatly distended with transudation and the bowels compressed, the note over the whole surface of the abdomen becomes absolutely dull. In cases of medium severity, when the patient lies on his back the fluid gravitates towards the lumbar regions, and the intestines float on its surface, so that the percussion note over the anterior surface of the abdomen is clear and tympanitic, expressing the presence of large air cavities beneath (bowels), while on either side, as we pass towards the lateral and posterior regions, there is dulness corresponding to the position of the ascitic fluid. If the patient lie on his left side, the right side will be the point towards which the air-containing intestines will float, whereas the fluid in the peritoneal cavity will gravitate towards the left. The change in the percussion note thus caused by alteration in the position of the patient is an indication of the presence of free fluid in the peritoneum, and is the more important seeing that it does not occur in the case of ovarian dropsy.

Ascites is either the result of increase in the blood pressure within the portal vein, due to cirrhosis of the liver, tuberculosis of the peritoneum, &c., or is merely a part of the general dropsical condition caused by cardiac or renal disease.

The differential diagnosis of ascites is often very difficult. It is not hard to distinguish it from meteorism, in which there is neither lateral dulness altering with position, nor the undulatory impulse mentioned on page 59, nor from an over-distended bladder, in which the dulness is centrally placed over the pubes. The pregnant uterus also is readily distinguished from ascites by the position of the dulness, and, above all, by the auscultation of the foetal heart. It is often, however, extremely difficult to distinguish ascites from ovarian cystic tumour. The main points of difference are given in the accompanying table.

ASCITES.	OVARIAN CYST.
<p><b>I. History.</b> No history of lateral development.</p>	<p>Tumour develops from one iliac fossa.</p>
<p><b>II. Inspection.</b> When patient lies on the back there is bulging at the flanks. If the ascites is considerable, the umbilicus is pressed outwards.</p>	<p>The greatest swelling is anterior, not in the flanks. Sometimes one side of the abdomen is more prominent than the other.</p>
<p><b>III. Percussion.</b> On percussion there is dulness in the flanks, and a clear note over the centre of the abdomen. Changes of position alter the line of dulness in the manner already described.</p>	<p>The dulness is central, the intestines giving a clear note at the sides.  Change of position does not alter the line of dulness.</p>
<p><b>IV. Aspiration.*</b> Ascitic fluid presents the following characters:—</p> <ol style="list-style-type: none"> <li>1. Specific gravity 1·010 to 1·015.</li> <li>2. Light straw colour.</li> <li>3. Coagulates spontaneously when exposed to the air.</li> <li>4. Does not contain paralbumen.</li> </ol>	<p>Ovarian fluid has the following characteristics:—</p> <ol style="list-style-type: none"> <li>1. Specific gravity 1·018 to 1·024.</li> <li>2. Amber coloured; often syrupy.</li> <li>3. Seldom or never coagulates spontaneously.</li> <li>4. Contain paralbumen.</li> </ol>

\* The microscopic differences between the two fluids cannot be said to be as yet clearly determined.

**Percussion of the Liver.**—A considerable portion of the anterior and upper surface of the liver lies in contact with the anterior wall of the abdomen, and consequently over this area the percussion note is more or less absolutely dull, expressing the presence of a solid organ underneath. This area is spoken of as the *absolute hepatic dulness*.

Above this the liver recedes from the chest-wall and becomes separated therefrom by a layer of lung of gradually increasing depth. In percussing from above downwards in the right parasternal line, the level at which the percussion note indicates that the subjacent air-space is being encroached upon and rendered shallower, corresponds (in the normal condition) to the highest point to which the liver reaches under the diaphragm, and at this level the deep or *relative hepatic dulness* commences.

The tympanitic resonance of the neighbouring abdominal organs, which contain air, enables us to mark out with considerable ease the lower border of hepatic dulness. The thinness of the lower edge of the liver necessitates that percussion should be made very lightly in order that we may avoid, as much as possible, the transmissions of the vibrations to parts in the vicinity. It is best practised, in this particular instance, by tapping very gently with the forefinger on an ivory pleximeter.

The *lower border of the liver* begins at the left, close to the apex of the heart, and passes diagonally downwards and towards the right, crossing the middle line at a point mid-way between the base of the ensiform cartilage and the umbilicus, and joining the margin of the ribs at an acute angle in the mammary line. From this point backwards to the axillary line the lower border corresponds pretty closely with the margin of the ribs. In many cases careful percussion will detect the presence of the gall-bladder, as a small rounded tumour projecting downwards from the edge of the liver.

The *upper border of absolute hepatic dulness* corresponds to the lower edge of the right lung, except in regard to the left lobe, where it passes imperceptibly into the cardiac dulness.

At the right border of the sternum it lies at the level of the sixth rib; in the mammillary line it corresponds to the upper border of the seventh rib, and in the axillary and scapular lines it reaches respectively the eighth and ninth ribs.

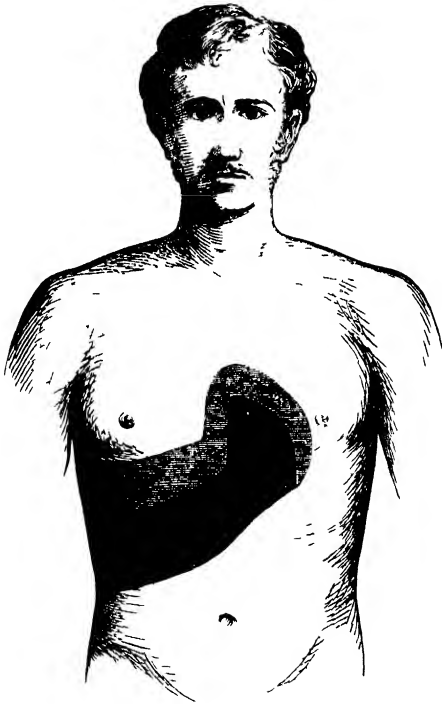


FIG. 9.—Cardiac and Hepatic Dulness.

- |                              |  |                              |
|------------------------------|--|------------------------------|
| 1. Relative hepatic dulness. |  | 3. Relative cardiac dulness  |
| 2. Absolute hepatic dulness. |  | 4. Absolute cardiac dulness. |

The upper limit of the deep or *relative hepatic dulness* lies about three inches above the absolute dulness. In the mammillary line the percussion note can usually be noticed to become dull about the fourth intercostal space, or fifth rib.

The position of the hepatic dulness, relative and absolute, is roughly indicated in the accompanying diagram (fig. 9).

The movements of respiration change the position of the liver. Deep inspiration depresses the lower edge considerably, while full inspiration permits of a corresponding elevation. But in addition to such alterations in the level at which the lower border of the liver stands, the respiratory movement affects the position of the upper border of the absolute dulness to a much greater extent. This latter alteration in hepatic dulness does not express so much a change in the position of the liver, as the rise and fall of the lower border of the right lung, and the extent of the complementary pleural space occupied by pulmonic tissue.

Changes in the position of the body also cause slight differences in the position of the liver, the organ gravitating towards the most dependent side.

The hepatic dulness may be greatly altered without any actual change in the size of the liver. Thus the colon, distended by air or by a coil of the small intestine, may be forced upwards between the surface of the liver and the abdominal parietes, thus preventing the true lower border from being found by percussion, and leading to an apparent diminution in the size of the liver. In pulmonary emphysema the right lung extends lower down than normally, and the upper border of absolute liver dulness is thus depressed. Again, the liver may be elevated abnormally within the concavity of the diaphragm, by reason of increased intra-abdominal pressure; a greater proportion of the organ will be thus overlapped by lung, and the absolute dulness diminished. If the two last conditions co-exist, all trace of absolute hepatic dulness may fail, and this may occur when the liver is of normal size.

From all this it follows that the extent of the absolute hepatic dulness is no safe guide to the size of this organ, and in all cases it is best to measure from the upper level of the deep or relative dulness to the lower border of the liver. Unfortunately, in some cases such measurements cannot be accurately made (right pleural effusion, pronounced ascites, &c.), but when percussion can determine the deep dulness, the position of

the lower border may in such cases usually be ascertained by palpation.

*Displacement of the liver* takes place more frequently downwards than upwards. The causes of displacement *downwards* are—(1) Emphysema of the lungs, when both hepatic lobes are equally depressed; (2) pleuritic effusion on the right side, which causes depression of the right lobe of the liver, with perhaps slight elevation of the left lobe; (3) right pneumo-thorax, producing the same conditions. To these may be added, as much rarer causes of downward displacement, various tumours of the mediastinum and of the diaphragm, and encapsuled peritoneal effusions between the diaphragm and the upper surface of the liver. The left lobe of the liver may be slightly depressed by large pericardial effusions, and by effusion of liquid or gas into the left pleural cavity.

*Displacement upwards* occurs less frequently, and to a less extent, than depression. It is occasioned by any condition which produces an increased pressure in the abdominal cavity—ascites, meteorism, ovarian cysts, &c.—and possibly also by cirrhosis of the right lung.

*Hepatic enlargement.*—The increase in size of the liver may be very marked. It may in rare cases rise as high as the second rib (Gerhardt), while its lower edge may reach to a point close to the symphysis pubis. The chief causes of great enlargement of this organ are hydatid tumours, carcinoma, and waxey disease. It is found to a less degree in hepatic congestion (as in mitral disease), occlusion of the bile ducts, fatty degeneration, and in certain cases of cirrhosis. (The alteration in the shape of the liver, caused by the practice of “tight-lacing,” may simulate actual enlargement.)

*Diminution in the size of the liver* occurs in the later stage of cirrhosis, and in acute yellow atrophy of the liver. The organ, as it becomes smaller, leaves the surface of the abdomen and retreats into the concavity of the diaphragm. Its place is occupied by small and large intestine, and, in consequence, all trace of hepatic dulness may disappear. This extreme diminu-

tion is met with in the latter disease, whilst in cirrhosis, when the liver is much contracted, the percussion of the lower border is prevented by the almost invariable presence of ascites at that advanced stage of the disease.

**Percussion of the Spleen.**—Placed obliquely in the cavity of the abdomen, the spleen lies with its upper and posterior extremity opposite the tenth dorsal vertebra, in the concavity of the diaphragm, and somewhat overlapped by the left lung. From this point the gland passes downwards and forwards to terminate slightly behind the extremity of the eleventh rib. Its upper and anterior border runs parallel with the ninth rib, while the posterior and lower border follows pretty closely the eleventh rib.

The percussion of the spleen is peculiarly important, because when the organ is but slightly enlarged, it is practically inaccessible to palpation. In consequence of the nearness of large air cavities in the stomach and colon, it is necessary to percuss very lightly in order to obtain the splenic dulness, and not allow the note to become obscured by reason of the tympanitic resonance of these organs.

The spleen is of variable size, and as age advances it atrophies so that a small area of dulness may be met with under physiological conditions.

Respiratory movements affect the position of the splenic dulness, deep inspiration depressing it and diminishing its size. When the patient lies on the right side the spleen tends to gravitate towards that direction, and the splenic dulness diminishes or disappears. When the spleen is not greatly enlarged, it is best to percuss it while the patient is in the erect position, with the left arm removed from the side.

The condition of the stomach has an important influence on the percussion of the spleen. If the fundus is greatly distended with food, it occasions a dull note on percussion in the neighbourhood of the spleen, in such a way as altogether to prevent the differentiation of the splenic dulness. On the other hand,

if the stomach be much distended with gas, it becomes difficult to determine, with any exactness, the limits of the splenic dulness, because of the tympanitic resonance of the gastric cavity which even a very light stroke can hardly fail to produce.

Conditions similar to those which cause upward displacement of the liver (p. 73) force the spleen in the same direction under the lower margin of the left lung ; and, in this case, as well as when the spleen is overlapped by emphysematous pulmonary tissue, all traces of splenic dulness may disappear.

The presence of ascitic fluid round the spleen will prevent its limits from being determined by percussion, and meteoric distension of the intestine with gas will cause a diminution in the size of the splenic dulness.

In pleuritic effusion and pneumothorax on the left side, as well as in pulmonary emphysema, the spleen is depressed, but in none of these conditions is it possible to define its limits by percussion.

*Enlargement of the spleen* is readily made out by means of percussion ; but this method does not give any clue to the cause of the tumefaction, as in such cases the gland enlarges uniformly in all directions. When it passes beyond the borders of the ribs, it is best recognised by means of palpation. The various forms of splenic tumour have been already referred to (page 61), and need not now be recapitulated.

**Percussion of the Kidneys.**—It is only in the rarest of cases that such a method of examination need be resorted to, to aid in forming a diagnosis, and this because (1) the most frequently occurring renal diseases are not accompanied by so great an amount of alteration in the dimensions of these organs as to be appreciable by percussion ; and (2) those cases of renal tumours where the tumour mass might be thus recognised are capable of far earlier and much more thorough investigation by palpation. Renal percussion may in fact be regarded as of little importance, except, perhaps, in the single instance of the diagnosis of float-



ing kidney, where the absence of the normal area of renal dulness on one side might confirm the opinion. But as undoubtedly in many cases the percussion of the kidneys can be carried out, the subject must not be entirely ignored here.

The percussion stroke must be very firm, and is best given by means of a hammer and ivory pleximeter.\*

The upper border of the renal dulness cannot be defined, as it passes imperceptibly into that of the liver or spleen, and the internal border lying next the spinal column is also incapable of definition. It is then chiefly the outer border, lying in the lumbar region, parallel to, and at about three finger-breadths distance from the spinal column, that can be marked out, while in some few cases the position of the lower border, close to the crest of the ilium, can also be ascertained.

The patient must be laid prone, the anterior surface of the abdomen being supported on cushions. The lower edges of the hepatic and splenic dulness in the scapular line on either side must first be marked out. Immediately below these levels the tympanitic note of the colon or of the stomach can be heard. If now we percuss inwards on either side towards the vertebral column, the renal dulness will be reached at the distance already indicated. The length of the renal dulness is usually from three to four inches, and occasionally, as I have already said, the lower border may also be defined by percussion.

[The *urinary bladder* does not enter the abdomen unless distended, and then it can readily be percussed out as a pear-shaped tumour lying in the middle line, and giving a dull note.]

**Percussion of the Stomach.**—When the stomach is filled with food, it is impossible to define its boundaries in any way by means of percussion; but when the cavity of the viscus is moderately distended with air, it gives on percussion a tym-

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\* In persons in whom there is great development of the subcutaneous adipose tissue, the renal dulness cannot be percussed out.

panitic note of so low a pitch and of so long duration, that it is readily distinguishable from the tympanitic notes obtained from the neighbouring hollow viscera, and this by reason of the greater size of the air cavity. Should, however, the distension of the stomach with gas increase beyond a certain point, the walls are also thrown into vibration, and a metallic ring results, which renders the definition of the gastric outlines more difficult.\*

The position of the stomach is such that its border can be satisfactorily defined only in one direction—i.e., in the line of the greater curvature; but the ascertaining of the position of this border is sufficient to enable us to say whether the viscus be enlarged or no.

It is best, first of all, to mark out the borders of the various other organs which surround the stomach, namely, the liver and spleen, as well as the position of the diaphragm. It is then found that the deep-pitched note of the stomach extends from the middle line to the left hypochondrium, neither passing to the right of the middle line nor below the level of the umbilicus. If either of those limits be overstepped at any point, the stomach is enlarged.

Over the *intestines* the percussion note is normally tympanitic, but is higher in pitch than over the stomach.

When the bowels become distended with liquid or solid contents, this note ceases to be heard.

Great distention with gas allows the walls of the intestines to pass also into vibration, thereby producing a metallic note.†

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\* I have already alluded (page 55) to the method of artificially distending the stomach with gas for purposes of diagnosis.

† Ziemssen has recently (*Deutsch. Arch.*, June, 1883) recommended, for diagnostic purposes, the artificial distension of the intestines with gas formed by a mixture within the rectum of bicarbonate of soda with a solution of tartaric acid. The form and position of the bowels can in this way be readily defined, and the exact seat of a stricture ascertained. For details the original must be consulted.

**Auscultation of the Abdominal Organs.**—Auscultation of the abdominal organs is rarely of value as an aid to diagnosis, if we except the auscultation of the uterus in pregnancy, which does not concern us here, and aneurisms of the abdominal aorta and its branches, which will be better considered in connection with the circulatory system.

## CHAPTER VI.

### Hæmopoietic System.

IN this chapter there fall to be considered the lymphatic system, the various ductless glands (spleen, thyroid, &c.), and the microscopic characters of the blood.

**1. The Lymphatic Vessels**, as well their capillary network as their larger trunks and the thoracic duct itself, undergo various pathological changes which, although in great measure belonging to the domain of surgery, come frequently under the observation of the physician. For convenience of description, the following order will be here followed :—

(a.) *Inflammation* of the lymphatics (Lymphangitis) depends almost invariably upon the absorption of some irritating (usually septic) material from the tissues. It sometimes follows some slight wound or sting, but often results from gonorrhœal, syphilitic, cancerous, or erysipelatous irritation. The superficial lymphatics show themselves as pale red lines passing up towards the glands, and the larger vessels may be felt as hard and knotted cords, and pressure over them is painful. There is usually some swelling of the surrounding tissues.

(b.) *Narrowing and Dilatation*.—Narrowing and obstruction of the lymphatic vessels may arise from the pressure of neighbouring tumours, from inflammation, thrombosis, &c. Dilatation (Lymphangiectasis) is commonly the result of this narrowing or obliteration, but it may arise also from disease of the

lymphatic glands preventing the flow of lymph. Sometimes this dilatation exists without giving rise to any symptom, but when the lymphatics affected are superficial, they may be readily felt. This occurs most frequently on the inner sides of thighs, on the scrotum, penis, and lower surface of the abdomen. Nodular vesicles, like sago grains, may then be felt lying under the skin, which sometimes spontaneously rupture, discharging lymph. They are to be distinguished from varicose veins by their position, form, and colour. Sometimes the enlarged lymphatic trunks take the form of a tumour (lymphangioma), usually in the tongue, cheek, or neck.

The *Thoracic Duct* itself is sometimes narrowed or occluded, most commonly as the result of the pressure of thoracic tumours (aneurisms, cancerous and tubercular disease of the mediastinal glands), and sometimes perhaps from inflammation of its coats or thrombosis. This obstruction is followed by dilatation of the duct (which may also arise from stasis in the large veins at the root of the neck, the result of heart disease), and dilatation, especially if it take place slowly, may give rise to no symptom. Very often, however, it is followed by marked wasting and anæmia, and sometimes by ascites, and even hydrothorax,—the fluid poured out in the pleural or peritoneal cavity resembling chyle.

(c.) *Rupture*.—The discharge of lymph (lymphorrhagia), follows every wound, and only becomes important when it flows from a large trunk, a condition which may be caused by wound, by ulceration, or by rupture following previous dilation. Its diagnosis can present no difficulty when the effusion of lymph takes place externally. Rupture is most frequent in the inguinal region. The lacteals sometimes rupture (the result of congestion from the pressure of a tumour), discharging their chyle into the peritoneal cavity, and usually giving rise to no symptom. Several cases are on record of rupture of the thoracic duct, caused by wound or by the bursting of a neighbouring abscess. The symptoms are obscure, but the presence of rupture of the duct might be diagnosed by the

flowing of chyle from the wound, or by the accumulation of that fluid in the pleural cavity.

Chylous urine, probably the result of rupture of the renal lymphatics, will be considered under the urinary system.

*Neoplasms* of the lymphatic vessels are so rare and so obscure as not to warrant their consideration in these pages.

**2. The Lymphatic Glands.**—Enlargement of the lymphatic glands may arise from various causes—

(a.) *Inflammatory.*—This may be the direct result of a blow, but more commonly arises from the absorption of irritating material from an abrasion. Such enlargement is common at the elbow, axilla, knee, and groin. The chain of femoral glands may enlarge as the result of a wound on the leg or foot, while irritation on the penis affects the inguinal chain (bubo). The glands at the back of the neck frequently swell where there exists any irritation of the scalp, and the cervical glands become enlarged in diphtheria and scarlatina.

(b.) *Scrofula.*—In strumous persons, particularly in childhood and early life, the glands of the neck enlarge, slowly suppurate, and caseate. In cases of *tabes mesenterica* scrofulous glands may be felt through the abdominal wall.

(c.) *Syphilis.*—The adenitis (almost invariably inguinal) which follows a soft chancre is called the “virulent bubo.” It comes on generally about a week after infection, is accompanied with acute pain, and ends in suppuration. The “multiple bubo” which follows the hard chancre after an interval of about three or four weeks, is as a rule painless; there is no suppuration, and the induration proceeds slowly from gland to gland in the inguinal chain until all are affected. They seldom exceed a walnut in size, are hard, and roll about freely under the finger. In the secondary stage of syphilis many of the lymph glands throughout the body are enlarged (particularly at the back of the neck, and the inside of the elbow).

(d.) *Cancer.*—In the neighbourhood of cancerous tumours

the lymphatic glands become enlarged and hard, and constitute an important element in diagnosis and prognosis. The most common example of this is the enlargement of the glands in the axilla, and above the clavicle, which follows carcinoma of the breast.

(e.) *Adenia or Hodgkin's Disease*.—This affection is characterised by an enlargement of the lymphatic glands throughout the body. They may attain to great size, but do not suppurate. There is marked anæmia, a diminished number of red blood corpuscles, but no increase of the white, and the spleen is generally enlarged. The cervical glands are usually the first to be affected.

(f.) *Leucocythæmia*.—In some cases of this disease there is a general enlargement of the lymphatic glands throughout the body, along with that of the spleen. It is readily distinguished from adenia by the increase in the white blood corpuscles which is present.

(g.) In the *Plague*, and also in *Anthrax*, *Glanders*, and *Farcy*, acute glandular swellings form in the axilla, groin, and neck.

**3. The Ductless Glands.**—*The Spleen* has been already considered under the abdominal system, and need not be again alluded to.

*The Thyroid*.—The very rare inflammation and simple enlargement of this gland, as well as the malignant and cystic growths which form in it, belong to the domain of surgery. There remain for consideration only two diseases.

(a.) *Bronchocele* or *Gôitre* is an endemic disease prevailing chiefly at the base of high mountains. The thyroid is enlarged, the whole or part of the gland being affected, and presses upon neighbouring parts, leading to difficulty of breathing and swallowing. *Gôitre* is often associated with mental deficiency (cretinism).

(b.) *Exophthalmic Gôitre*—*Graves' Disease*.—In this affection the swelling of the thyroid (usually the right lobe) is of a

vascular character—soft, elastic, and pulsating. It is accompanied with marked pulsation in the vessels of the head and neck, palpitation of the heart, prominence of the eyeballs, and other nervous symptoms, and is most common in females below middle age.

*Thymus*.—Diseases of the thymus hardly come within the limits of possible diagnosis, except those lymphatic and malignant tumours of the anterior mediastium, which have their starting-point in that gland.

## BLOOD.

The examination of the blood has of late years attained to such importance as an aid to diagnosis, and as a guide in the subsequent treatment of many diseases, that the various processes employed demand a somewhat minute description. Limiting ourselves to those methods which may be truly called *clinical*—i.e., which can be carried out at the bedside of the patient—and therefore excluding the minute chemical analysis which can only be performed in the laboratory, and for which the comparative infrequency of blood-letting gives but few opportunities during the lifetime of the patient, we shall find the lines of investigation to be mainly three—

1. Microscopic examination of the blood.
2. Enumeration of corpuscles it contains.
3. Estimation of the hæmoglobin.

**1. Microscopic Examination of Blood.**—To obtain a drop of blood for examination, the end of the patient's finger must be carefully cleansed, and a prick made with a needle. The finger must not be squeezed or compressed in any way, as this tends to alter the proportion of serum and corpuscles which the blood contains. The drop of blood so obtained should be received on a clean microscopic slide, the cover-glass applied, and the microscopic examination made as speedily as possible. The



pathological changes usually met with may be divided into three classes—

- (a.) Alterations in the form of the normal constituents of the blood.
- (b.) Variations in their number.
- (c.) The presence of foreign substances.

(a.) *Alterations in the form of the Normal Constituents of the Blood.*—In anæmia, particularly in the pernicious variety of that disease, the red blood corpuscles vary much in size, some being larger and some smaller than normal, and all having a pale appearance, due to loss of hæmoglobin. In some instances they tend to assume curious and irregular shapes of various kinds. The changes in the colourless corpuscles are not so distinctive, but not infrequently they may be found, in case of anæmia and leucocythæmia, to be abnormally refractive, and to contain granules resembling oil-globules. In various cachetic conditions the protoplasmic granules, which are to be found occasionally in normal blood, become much increased in number, and adhere together so as to form larger or smaller masses.

(b.) *Variations in the Number of the Blood Corpuscles.*—Whilst in many cachetic conditions, as, for example, in phthisis, the number of leucocytes in the blood may be above the normal (leucytosis), it is in leucocythæmia that this increase is by far most observable, and by means of the microscopic examination of the blood alone, a diagnosis may be arrived at. The number of leucocytes present in the field may be very great, even in pronounced cases surpassing that of the red corpuscles. In the lymphatic variety of the disease the size of the leucocytes is very much that of ordinary lymph corpuscles, and they contain as a rule but one nucleus, while in splenic leucocythæmia they are larger in size, and usually possess more than one nucleus. The diminution in the number of red corpuscles in anæmia, though not so striking as the increase of leucocytes just mentioned, is of considerable clinical interest.

(c.) *The presence of Foreign Substances.*—Among these may be mentioned, in the first place, the dark brown or black

pigment granules, which are sometimes found in considerable numbers in the blood after malarious fever. This condition is spoken of as *melanæmia*.

More important is the presence of the various micro-organisms which appear in the blood in certain diseases. I do not think it necessary here to refer to more than one or two of these forms, those namely in which the diagnosis rests much or mainly upon the evidence supplied by the microscopic examination of the blood.

*Bacillus Anthracis*.—This form of organism, is found in the blood of persons suffering from the disease which is known under the different names of anthrax, woolsorter's disease, splenic fever, and malignant pustule. The blood is found to be filled with short rods, somewhat exceeding in length the diameter of a red blood corpuscle, which, as the disease advances, become changed into long thread-like filaments. These forms are usually to be made out with very considerable ease, but they will be rendered more distinct if the blood be allowed to dry on the cover-glass, and be then stained with methyl-violet, or other appropriate reagent.



FIG. 10.—*Bacillus Anthracis*.  
(After Koch.)

*Spirochæte Obermeierii*.—This spirillum, which is found in the blood of patients suffering from recurrent fever, possesses a very characteristic appearance.

On examining a specimen of such blood with the microscope, it will be seen that, moving actively among the blood corpuscles, are a number of thread-

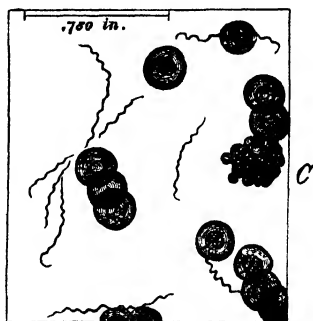


FIG. 11.—*Spirochæte Obermeierii*.  
(After Carter.)

like organisms. Their length is usually three or four times greater than the diameter of a red corpuscle, and in shape they resemble a cork-screw. Their movements are sometimes so powerful as to move the blood corpuscles with which they may come in contact in their course.

Besides these, there are, as I have said, a considerable number of micro-organisms met with in the blood of different diseases, but although their form is usually sufficiently distinctive, yet their recognition by means of the microscope cannot

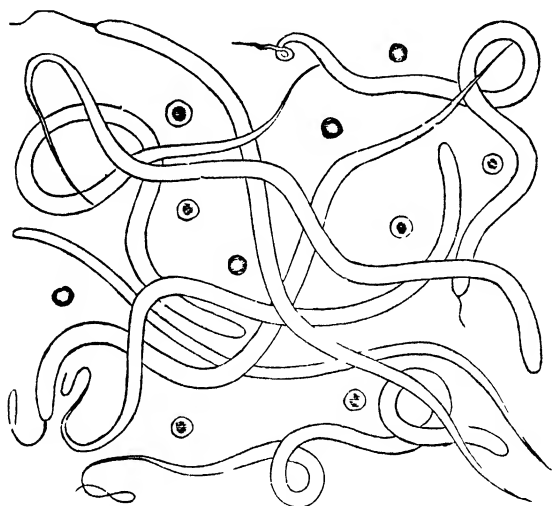


FIG. 12.—*Filaria Sanguinis Hominis*. (Roberts)

be held to have as yet assumed sufficient practical diagnostic importance to justify any detailed description in these pages.

Mention must, however, be made of a much more highly developed parasite which is occasionally met with.

*Filaria sanguinis hominis* is the name which Lewis has given to a nematode worm which is found in the blood of persons suffering from chyluria, certain forms of endemic hæmaturia, elephantiasis, lymphoid affections, &c. These *filariæ* are exceedingly minute, and are the sexually immature form of

*Filaria Bancrofti*, a long thread-like worm which has been found in lymphatic abscesses.

**2. Enumeration of the Corpuscles in the Blood.**—This method of investigation, which was first attempted by Vierordt, has been followed out by numerous observers, each of whom has described a special form of apparatus for the purpose.

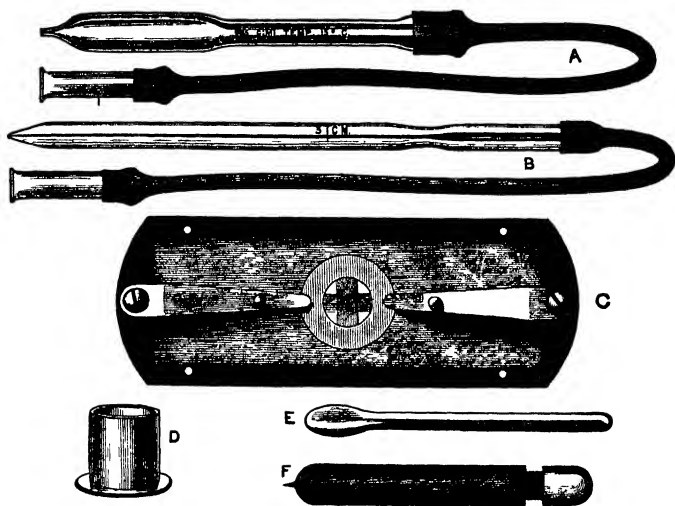


FIG. 13.—Gowers's Apparatus for counting Blood Corpuscles

A, Pipette for measuring the diluting solution, B, Capillary tube for measuring blood; C, Cell, with divisions in the floor, mounted on a slide to which springs are fixed to secure the cover glass, D, Vessel in which dilution is made, E, Spud for mixing the blood and solution, F, Guarded needle

While the most recent modifications of Malassez's instrument, or the apparatus of Abbé-Zeiss, probably give the most rigidly accurate results, I shall here describe the instrument designed by Dr. Gowers,\* which is exceedingly handy, and is the form most frequently used in this country.

\* Made by Hawksley, 300 Oxford Street, London. A full description of the instrument will be found in *The Practitioner* for 1878.

Gowers's *Hæmacytometer* consists of—(1) a small pipette, (A in fig. 12), which, when filled to the mark on its stem, contains 995 cubic millimetres; (2) a capillary tube (B) marked to contain five cubic millimetres (each of these is furnished with an india-rubber tube and mouthpiece); (3) a small glass jar (D), in which the blood is to be diluted; (4) a glass stirrer (E) for mixing the blood and the solution in the jar; (5) a brass stage-plate (C) carrying a glass slip, on which is a cell  $\frac{1}{10}$ th of a millimetre deep. The bottom of this is divided by intersecting lines  $\frac{1}{10}$ th millimetre squares. Upon the top of the cell rests the cover-glass, kept in position by means of two springs.

The diluting solution may vary. One which answers well consists of a solution of sulphate of soda, 26 grains in one ounce of distilled water, to which is to be added 15 drops of strong acetic acid.

When an observation is to be made, 995 cubic millimetres of the solution are measured by means of the pipette, and placed in the mixing jar. The patient's finger is now to be pierced with the spear-pointed needle supplied along with the instrument, and a drop of blood obtained without any squeezing of the finger, which, as I have already said, alters the proportion of serum and corpuscles in the blood. Five cubic millimetres of this blood are drawn into the capillary tube, and then blown into the diluting solution in the jar, any superfluous blood being previously removed from the point of the pipette by means of a soft cloth. The two fluids are well mixed by rotating the stirrer between the finger and thumb, after which a small drop is placed in the centre of the cell, and the cover-glass applied. The drop must lie in the *middle* of the cell, and must not touch its sides. The slip is now placed upon the stage of a microscope, and the lens focussed for the squares, which are marked out by lines drawn on the floor of the cell. In a few minutes the corpuscles sink through the drop, and come to rest on these squares. The number in ten squares is then counted, and this multiplied by 10,000 gives the number in a cubic millimetre of blood.\*

\* Probably it would conduce to greater accuracy if the mixture of

In counting the white corpuscles, it is well to observe the number of squares in a field and then the number of white corpuscles in a series of fields—raising the focus until they appear like bright points on account of their refractive power.

The most recent calculations show the number of coloured corpuscles given by Vierordt—5,000,000 per cubic millimetre—to be approximately correct. The average number in two squares is 100, and Gowers proposes to take this quantity as the “hæmic unit.” This is a very convenient way of calculating, for the number of corpuscles per “hæmic unit,”—*i.e.*, in two squares (counting ten or twenty squares, and taking the mean), expresses the percentage proportion of the corpuscles to that of health. The number of white corpuscles in ten or twenty squares is easily counted, and the proportion of white to red ascertained. The normal *maximum* of white per two squares (hæmic unit) is .3.

In all conditions of anæmia and cachexia the number of red corpuscles undergoes diminution, and by making an estimation from time to time we can obtain most valuable and trustworthy indications regarding the progress of the malady and the effect of treatment.

**3. Estimation of Hæmoglobin.**—Various instruments have been devised for this purpose by Malassez and others. That of Dr. Gowers,\* which I prefer both on account of its simplicity and the accuracy of the results which it gives, consists of two glass tubes of the same diameter, one of which contains a

blood and solution were accomplished by means of Potain’s “Mélangeur,” and if a special cover-glass ground absolutely level were supplied along with this apparatus; and, further, if this cover-glass were fixed in such a manner to the slip that it might be steadily lowered on to the drop by means of a rack movement. All of these arrangements have been adopted by Malassez in the newest model of his instrument.

\* Described in full by Dr. Gowers in the “Transactions of the Clinical Society of London,” vol. xii. 1879.

standard colour-solution \* (glycerine carefully tinted by means of carmine and picrocarminate of ammonia), while the other, in which the blood to be tested is to be diluted, is graduated so that 100 degrees = two cubic centimetres. There is also a capillary pipette graduated to hold twenty cubic millimetres, a bottle with a pipette-stopper to contain distilled water, and a guarded needle to prick the finger.

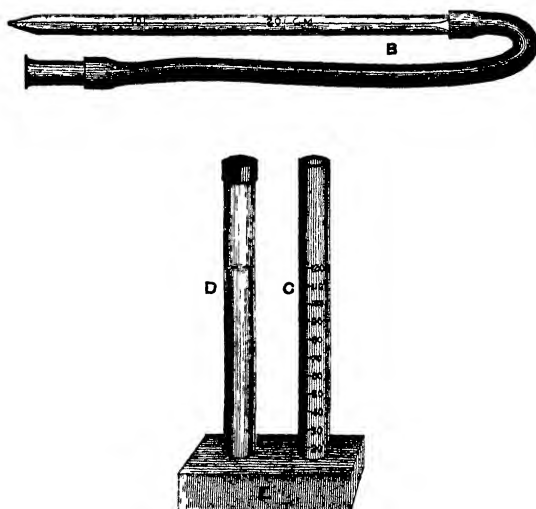


FIG. 14 —Gowers's Apparatus for estimating Hæmoglobin.

The method of using this instrument is as follows. The two tubes (c and d, fig. 14) having been placed upright in the small wooden stand (E) supplied for the purpose, a few drops of distilled water are placed in the bottom of the graduated tube. The blood having been obtained from a prick in the manner already described, twenty cubic millimetres of the blood are measured off by means of the pipette (B), and injected into the

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\* The tint of this standard solution corresponds exactly to that of a dilution of twenty cubic millimetres of blood with 1980 cubic millimetres of distilled water—*i.e.*, a dilution of one in a hundred.

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distilled water in the graduated tube, which must then be quickly shaken to ensure thorough mixture. More distilled water must now be added drop by drop until the tint of the diluted blood is the same as that of the standard. The degree of dilution, as indicated by the graduation, expresses the amount of hæmoglobin as compared with that of the standard, and as this is a dilution of one hundred, the degrees of dilution required to obtain the same tint represent the percentage proportion of the hæmoglobin to that of normal blood.\*

If the corpuscular richness of the blood is ascertained by means of the hæmacytometer, we are able to compare this with the amount of hæmoglobin in a very instructive manner. Thus, a fraction, of which the numerator is the percentage of hæmoglobin, and the denominator the percentage of corpuscles, will express the average value of each corpuscle.

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\* Thus, if the tints are identical when the dilution has reached eighty degrees, the blood contains only eighty per cent. of the normal quantity of hæmoglobin.



## CHAPTER VII.

### Circulatory System.

#### SUBJECTIVE PHENOMENA.

BEFORE addressing ourselves to the physical examination of the heart, there meet us for consideration certain symptoms of a more or less subjective kind.

1. *Pain*.—In anæmic persons, and particularly in women suffering from uterine disease, from chlorosis, or from nervous affections, pain over the region of the heart is frequently complained of, and true cardiac pain may likewise be simulated by neuralgia in the chest wall. In heart disease of any kind, and in particular in fatty degeneration associated with gout, pain may be a more or less prominent symptom. In its most pronounced form—angina pectoris—it comes on in recurring attacks of short duration, but of extreme severity. The first of the attacks usually occurs when the patient is making some exertion. The chest feels as if held in a vice, the pain, which is always severe, and which may be of the most intense character, radiates from the heart to the shoulders, and down the left arm, or down both arms to the wrist, breathing almost ceases, the countenance sometimes becomes livid, and consciousness may be lost. In other cases, the attacks may be very frequent, several in a day, or even one after the slightest exertion of body or of mind. The attack passes off as rapidly as it came on, and the patient may be free from its repetition

for months or years. The pain of aortic aneurism is more lancinating and more continuous than that of angina pectoris.

2. *Palpitation*.—The abnormal perception of excited pulsation in the heart or aorta is very frequently due to mental excitement, to dyspepsia, flatulence, anæmia, or nervous debility, or to the action of tea or coffee, and is often met with in cases of exophthalmic gôitre. It also occurs as a result of organic disease of the heart, and in such cases it will be found to be aggravated by exertion. The palpitation may be objective—i.e., the physician may himself recognise the excited action of the heart; or it may be merely subjective—the patient complaining of that sensation without there being the slightest evidence from physical examination of any alteration in the strength of the cardiac pulsations. Derangements of cardiac rhythm will be noticed hereafter.

3. *Fainting (syncope)*, which is primarily due to failure of the heart's action, is usually ushered in by a train of symptoms of which the chief are—pallor of the face, chilliness, cold perspiration, a feeling of weakness, of sinking in the epigastrium, and of sickness, pulse small and rapid, or slow and irregular, dimness of vision, ringing in the ears, and gradually increasing unconsciousness. Syncope may be due to organic disease of the heart, to nervous disturbance of the cardiac action (central or reflex), to intense mental emotion (hysteria), to deficiency of the blood supply to the heart muscle, or to want of blood in its cavities. It is of short duration, seldom lasting more than half-an-hour, and can in this way be distinguished from shock. Syncope may be simulated by apoplexy and by epilepsy, but the absence of paralysis and of muscular spasms enables a diagnosis to be readily arrived at. The action of alcohol in large amount, and of certain poisons, may produce a state closely resembling syncope, but the state of the pulse is here a reliable guide, unless indeed the poison used act as a cardiac depressant, when the diagnosis may be a matter of very great difficulty.

4. *Dyspnœa*.—The phenomena of dyspnœa, or difficulty of breathing, will be described under the head of the respiratory

system. It is, however, necessary to note here that this condition is common in valvular disease of the heart, due to the fact that the pulmonary circulation is interfered with. The dyspnœa may be constantly present, or may only show itself after exertion.

## CHAPTER VIII.

### Circulatory System—(*continued*).

#### INSPECTION.

THE cardiac or *præcordial region* corresponds to the lower part of the anterior mediastinum. It may be said to extend vertically from the second interspace to the sixth cartilage, and transversely from the apex-beat to a point about three-quarters of an inch to the right of the sternum.

The region so marked out overlies the heart, and the margins of both lungs which overlap it. More deeply still lie the organs contained in the posterior mediastinum.

In this chapter will be considered: (1) the form and appearance of the *præcordia*, and (2) the various pulsatory movements which show themselves on the walls of the thorax.

**Præcordia.**—A slight degree of bulging of the thoracic wall in the cardiac region is more readily detected by simple inspection than by means of measurement. It may be the result of curvature of the spinal column anteriorly and to the left, but is more commonly caused by cardiac hypertrophy, pericardial effusion, aneurismal and other tumours adjacent to the heart, or circumscribed pleuritic effusions. When effusion takes place into the pericardial sac, the intercostal spaces widen, they become raised to the level of the ribs, and ultimately may even protrude beyond them.

Depression of the præcordial region, on the other hand, may occur during the absorption of a pericardial effusion, and may remain permanently if extensive adhesions have been formed.

Bulgings caused by aneurisms lie almost without exception above the fourth rib.

### **Pulsations.**

1. *The Cardiac Impulse.*—In health the apex-beat of the heart is found in the fifth left interspace, about an inch and a-half or two inches from the left margin of the sternum, and its area does not exceed a square inch in extent. In childhood, however, and in persons who have a short and wide thorax, it may stand as high as the fourth interspace, and may be thrown somewhat farther to the left; whilst in old age, and in individuals whose thorax is very long and narrow, the cardiac impulse is depressed to the sixth interspace.

While natural breathing does not affect its position, deep inspiration and expiration cause respectively depression and elevation of the apex-beat.

When the patient lies on either side, the apex-beat is deflected in a corresponding direction. This alteration is more marked towards the left.

The cardiac impulse does not always make itself visible on the chest wall. This is usually due to great development of fat or muscle, and in such cases we can by palpation almost always fix the position of the apex-beat.

[Pathological changes in the position of the apex-beat will be considered under the head of Palpation.]

*Systolic indrawing* of the thoracic wall is of two varieties—(1) a recession, which is exactly synchronous with each ventricular systole; and (2) one which immediately succeeds the retirement of the apex of the heart from the chest wall.

The former variety is sometimes met with in healthy persons (particularly children) in whom the chest walls are unusually thin. It occurs in the third and fourth interspaces, and is simply the result of that recession of the base of the heart which

is synchronous with the forward movement of the ventricles. The chest walls are sucked inwards (or rather forced inwards by atmospheric pressure) to prevent the formation of a partial vacuum behind them which would otherwise take place.

The second form is seen at the apex, and is, according to Skoda, pathognomonic of adherent pericardium.\* If the adhesion be extensive (and particularly if the parietal layer be adherent anterior to the wall of the chest and posteriorly to the vertebral column) not merely the intercostal space but even the ribs may be drawn inwards, following the apex of the heart.

(2.) *Pulsation at the root of the neck* may be arterial or venous.

Pulsation in the carotid arteries becomes evident whenever the heart's action is increased in strength (as after great bodily exertion, or from mental excitement), but in its most pronounced form such pulsation is seen in cases of hypertrophy of the left ventricle, along with aortic incompetence. Pulsation in the jugular fossa when well marked usually points to simple or aneurismal dilatation of the aorta.

Swelling of the jugular veins is found in cases in which there is some obstruction to the return of blood to the heart, whether that obstacle be situated in the systemic or pulmonary circulation. If from any cause the right ventricle be unable to empty itself completely of blood, it becomes engorged, and, reacting on the right auricle, causes its dilatation; while the auricle so dilated in its turn retards the flow of blood through the jugular veins, which then exhibit distension. The same effect will, of course, be produced by any obstruction to the return of blood to the heart, whether seated in the lungs themselves, or at the mitral orifice, or the valves which close it.

This distension is necessarily accompanied with more or less

\* This sign, is not, however, invariably present in such cases, and though adherent pericardium is by far its most common cause, yet it may occur in cases in which the normal movements of the heart are otherwise hindered.

pulsation in the vein, the blood being only able to reach the heart during inspiration. This, however, is not the only pulsatory movement which the veins in this region exhibit when they are in a state of distension. The systole of the right ventricle causes a vibration which passes through the tightly-stretched right auriculo-ventricular valve, and the thrill thus communicated to the blood in the dilated auricle is thence transmitted to the jugular veins. In this case the tricuspid valve and the valves at the mouth of the jugular veins are competent, and there is, therefore, no backward flow of blood into auricle or vein; it is simply the impulse which is transmitted. This we can readily satisfy ourselves of by compressing the right jugular vein high up in the neck, and then if the contents of the lower part of the vessel be pressed out, the vein will not fill again from below, since no valvular incompetency exists.

When, however, the tricuspid valve is incompetent, or when the valves in the jugular vein cease to close the lumen of that vessel (either from destruction of its valves, or from extreme dilatation of the vein preventing the valves from doing their duty), the vein when so emptied will be seen to fill from below with regular pulsations corresponding to those of the right ventricle.

Thus is formed the "venous pulse," one of the most important signs of tricuspid incompetence.

Jugular pulsation may occasionally be præ systolic in rhythm, the movement resulting from the transmission of the impulse of the auricular systole into the vein.

Sudden collapse of the jugular veins during the ventricular diastole has been shown by Friedreich to be a sign of pericardial adhesion.

3. *Epigastric Pulsation* may be conveniently divided into two groups—(1) that which is synchronous with the ventricular systole, and (2) that which follows the systole after a slight, but appreciable, delay.

(a.) *Synchronous with the Ventricular Systole*.—When the

right ventricle is hypertrophied and dilated, it may frequently be felt to pulsate in the epigastrium, and any condition which depresses the diaphragm, or forces the heart towards the right, may give rise to this pulsation.\*

The liver may also pulsate in the epigastrium, but if the impulse is exactly systolic in rhythm, it can only be occasioned by direct transmission from the adjacent right ventricle.

(b.) *Delayed Epigastric Pulsation*—i.e., that which succeeds the ventricular systole after an appreciable interval,—may be due to the transmitted impulse of the abdominal aorta. The pulsation is then somewhat to the left of the middle line; it extends downwards towards the umbilicus, and is not diffused laterally. It may be conducted to the parietes by means of tumours, or through the overlying liver. The pulsation may be due to an aneurism on the abdominal aorta or one of its branches, when it will have a distensile character.

The venous pulsation which has been already noticed as occurring in cases of incompetence of the tricuspid valve is not limited to the jugular veins. It also takes place in the inferior vena cava, and the pulsation may in this way be communicated to the liver. If the hepatic veins be likewise affected, the pulsation in the liver becomes not merely heaving but distensile.†

In all these conditions the pulsation follows the apex-beat after a slight interval of time. The delay can be best appreciated by fixing with wax, over each pulsating point, a bristle carrying a small flag.

Systolic indrawing of the epigastrium occurs rarely, and is caused by extensive pericardial adhesions.

4. *Arterial Pulsations on the Thoracic Wall.* — Aortic aneurisms frequently give rise to visible pulsation in the upper part of the thorax, above the third rib. Such pulsation is

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\* Rosenstein has proposed to call this pulsation "parepigastric," as it lies rather at the border of the left ribs than in the centre of the epigastrium.

† To discriminate the various epigastric pulsations mentioned requires the use of palpation as well as inspection, but to preserve the continuity of the subject, they are all grouped together in this chapter.



systolic in rhythm, being as nearly as possible synchronous with the ventricular systole. If the ascending portion of the arch be involved, the pulsation usually lies to the right of the sternum ; if the transverse portion be the seat of the disease, the pulsation is more in the middle line ; and if the aneurism affect chiefly the descending part of the arch, the pulsation lies to the left side of the sternum. Aneurisms of the innominate and subclavian arteries also give rise to visible pulsation in the walls of the thorax. Systolic pulsation at the second left interspace is sometimes communicated to the surface from the subjacent pulmonary artery in cases of retraction of the borders of the lungs.

A diastolic impulse is sometimes to be seen and felt over the seat of the aortic and pulmonary valves (second right and second left intercostal spaces) in very emaciated persons, especially when the borders of the lungs have become retracted

5. *Capillary Pulsation* can occasionally be observed on the cheeks, beneath the nails, or in the line of congestion caused by drawing a sharp point, such as that of a pencil, over the skin, and though sometimes occurring independently of that cause, is usually due to incompetence of the aortic valve with hypertrophy of the left ventricle.

## CHAPTER IX.

### Circulatory System.

#### PALPATION.

THE skilled application of the hand to the cardiac region gives important information regarding the heart, which inspection alone is not fitted to communicate, and confirms much that inspection has already indicated. Palpation deals chiefly with the phenomena of the cardiac impulse, and with the various thrills which may occur in connection with the heart's action.

#### CARDIAC IMPULSE.

The apex-beat has already been spoken of in the last chapter in its normal condition. We now consider the various changes which it may undergo in disease—alterations in position, in strength, and in extent—remembering that in health the impulse lies between the fifth and sixth ribs, about one and a-half or two inches to the left of the sternum; that while ordinary respiration does not affect its position, it is depressed and elevated to a very slight extent by deep inspiration and expiration; and that when the patient lies on the left side it is slightly displaced outwards.

#### Alterations in the position of the Apex-beat.

##### 1. *Vertically.*

The height depends simply on the level of the diaphragm.\*

\* With the exception of cases of pericardial effusion, when the diaphragm may be depressed and the apex-beat simultaneously raised.

Pulmonary emphysema and spasmodic contraction of the diaphragm cause a general depression of the diaphragm, while collections of liquid or of gas in one pleural cavity or in the pericardial sac, an increase of the weight of the heart (particularly from hypertrophy of the left ventricle), or the presence of tumours in the neighbourhood of that organ, produce a local depression in the diaphragm, and each of these conditions finds expression in a lowered position of the apex-beat.

On the other hand, if the diaphragm be raised owing to cirrhosis of one lung, or contraction following the absorption of a pleural effusion, or by reason of increased abdominal pressure, the result of tumours, ascites, meteorism, &c., the apex-beat will be correspondingly elevated.

## 2. *Laterally.*

(a.) *To the right.*—Hypertrophy and dilatation of the right ventricle displace the apex-beat from its normal position towards the right. In slighter cases the pulsation is greatest in the epigastrium, but in those which are more marked, the apex, or at any rate that portion of the heart-wall which represents the apex, beats under the right edge of the sternum, or even farther to the right. Effusions in the left pleural cavity also push the heart over to the right side; and on the absorption of the exudation, the heart returns again to its normal position, provided that it has not become bound down by adhesions in its abnormal situation. The apex-beat may be found on the right side of the thorax in cases of congenital transposition of the viscera.

(b.) *To the left.*—Hypertrophy and dilatation of the left ventricle not only depress the apex-beat, but also move it considerably to the left. In cirrhosis of the left lung the heart follows the contracting lung towards the left, and effusions into the right pleural cavity also produce a movement of the apex-beat in this direction, when they are of considerable amount.

**Strength of the Apex-beat** varies much even in healthy individuals, owing to varieties in the thickness of the chest-

wall, in the width of the intercostal spaces, and in the extent to which the apex is overlapped by pulmonary tissue. Pathologically, the differences are still more apparent.

Diminished force of the cardiac impulse, even to the extent of being imperceptible to the finger, may be due to—

1. *Intrinsic causes*.—These include abnormal innervation, fatty degeneration of muscular fibre of the heart, myocarditis, and that degeneration of the heart muscle which follows hyperpyrexia of long duration, and lastly, deficiency of proper blood supply and interference with the circulation.

2. *Extrinsic causes*.—When the visceral and parietal layers of the pericardium become adherent, the movements of the apex of the heart are so interfered with, that all cardiac impulse may be lost. Effusions of fluid or gas into the pericardium or into the left pleural cavity, as well as intervention of emphysematous lung tissue between the heart and the thoracic wall, all tend to weaken the apex-beat.

*Increase in force* of the cardiac impulse may be of neurotic origin; it may be the result of fever, of endocarditis, of pericarditis or of any condition which is more than usually favourable to the conduction of the impulse, but by far its most common cause is hypertrophy of the heart. The heaving impulse which results from hypertrophy of the left ventricle, is much more easily detected than that which hypertrophy of the right ventricle occasions, as the latter has its point of maximum intensity behind the sternum.

The *rhythm* of the cardiac pulsations will be noticed in the remarks made on the arterial pulse.

**The Extent of the Impulse.**—Normally, the apex-beat is not perceptible over more than an area of about an inch in breadth, and is limited to the fifth intercostal space. When the pulsation extends much beyond such limits, it is abnormal. In disease the apex-beat not unfrequently becomes diffused over a considerable area, and this may result from increased action of a normal heart (medication, excitement, &c.), from cardiac hypertrophy,

from the application of an unusually extensive area of the heart to the thoracic walls (retraction of the lungs), or merely from great thinness of the chest-wall.

**Double Apex-beat.** — Independent, non-synchronous contraction of the ventricles has been met with in a few rare cases (Charcelay, Leyden, Roy, &c.), where the systole of the right ventricle caused a pulsation in the jugular veins which alternated with that in the carotid arteries.

**Thrills** may be felt by the hand applied over the cardiac region, and these are of two kinds—

1. *Endocardial thrills* are caused by the vibrating eddies which ensue when the blood current is forced through a small opening into a wider space. These conditions are satisfied in cases of stenosis of one of the orifices of the heart, or incompetence of a valve, when at the same time the blood current is sufficiently rapid. The pathological condition which gives rise to the thrill is indicated by the seat of greatest intensity, and the time in relation to the various phenomena of cardiac action.

Thrills in the mitral area (a circle with a radius of one inch round the apex-beat) are systolic or præ systolic according as they are produced by incompetence of the mitral valve or stenosis of the orifice which that valve covers. Thrills over the second right costal cartilage arise from aortic stenosis or incompetence; in the former case being systolic, and in the latter diastolic in rhythm. Præ systolic and systolic thrills in the tricuspid area indicate respectively stenosis of the orifice and incompetence of the valve. Very rarely a systolic thrill is felt over the pulmonary artery, denoting stenosis of the pulmonary orifice, or a diastolic, indicative of regurgitation.

2. *Pericardial friction fremitus* caused by the rubbing during the heart's action of the two pleural surfaces, which have been rendered rough and uneven by the effusion of lymph is more readily detected by auscultation than by palpation.

## CHAPTER X.

### Circulatory System—(continued).

#### PERCUSSION.

THE heart, lying in the thoracic cavity, has, in its normal condition, the following relations to the anterior wall of the chest :—

The *right border*, formed almost entirely by the right auricle, stretches in a slightly curved manner from the second right intercostal space downwards and outwards to the cartilage of the fifth rib on the same side, just at its junction with the sternum. The *left border*, formed by the left ventricle, reaches from the second intercostal space on the left side downwards and outwards to a point about half-an-inch outside of the apex-beat. The *lower border* corresponds pretty exactly to a line joining the sternal end of the fifth right costal cartilage with the apex-beat. The highest portion of the heart, formed by the appendix of the left auricle, reaches a level which may be indicated by a line joining the lower border of the sternal ends of the second pair of costal cartilages.

To percuss the heart, it is best to have the patient recumbent. No strong percussion is, as a rule, required, and this is specially the case with regard to the absolute dulness, where the tap should be light. In addition to the note elicited, the sense of resistance perceived during percussion is of great value.

The greater portion of the heart is separated from the chest

wall by the overlapping lung, and therefore it is only the small uncovered part, consisting of the right ventricle, which yields on percussion an absolutely dull note. This area of *absolute cardiac dulness* is readily mapped out. Its right border extends along the right margin of the sternum (or along the mid-sternal line in some cases) from the level of the fourth to that of the sixth costal cartilage. The left border is an irregular line stretching from the upper end of the right border to the apex-beat. The lower edge cannot be defined by percussion, because at this point the cardiac merges into the hepatic dulness. Its position, can, however, be obtained with approximate accuracy by drawing a line from the upper limit of absolute hepatic dulness on the right side to the apex-beat. The area thus formed is roughly quadrilateral in shape; and not only do its extent and position vary with each respiration, but they are also affected by the position of the person, and further, by the constant changes of the diaphragm in respiration, necessarily producing corresponding changes in the lie of the heart. The condition of the margins of the lungs (as in emphysema) have also an important influence on the size and position of the area of absolute cardiac dulness. The variations to which this area is thus liable greatly diminish the value of its condition as indicative of disease.

The region of absolute dulness is *increased* in—

(1.) *Hypertrophy and Dilatation of the Left Ventricle*, when the increase takes place chiefly downwards and to the left.

(2.) *Hypertrophy and Dilatation of the Right Ventricle*.—In this case the left margin is little interfered with, while the right is thrown outwards towards the right.

(3.) *Serous Effusion into the Pericardium*.—The dulness here takes a pyramidal form, being limited by the pericardial sac, with the base downwards, resting on the diaphragm.

(4.) Increase of absolute cardiac dulness may be simulated by various pathological conditions of the neighbouring organs, such as infiltration of the margins of the lungs, pleuritic effusions, &c.

It must be borne in mind, however, that these various diseased conditions of the heart and its investing sac may be present without giving rise to any appreciable changes in the absolute cardiac dulness. The area of absolute dulness is diminished, or entirely lost, in—

(1.) *Left pneumo-thorax*, where the collection of gas in the left pleural cavity is so great as to force the heart to the right. The area of dulness is usually, in such cases, still to be detected to the right of the sternum; but it is much diminished in size.

(2.) *Emphysema*, when well pronounced, entirely does away with absolute cardiac dulness, the margins of the lungs approaching so near to one another as to overlap the heart completely. In slighter cases, the area is only diminished in size.

(3.) In the rare cases in which free gas is found in the pericardium (pneumo-pericardium), percussion elicits a clear note over the whole cardiac area.

If, then, as has been said, the indications regarding the heart itself obtained by examination of the area of absolute cardiac dulness may be vitiated by various pulmonary conditions, we must seek to obtain information from the percussion of that portion of the heart which is covered by a more or less thick layer of lung.

In percussing the thorax, as we pass towards the cardiac area, the note, which is at first purely pulmonary in character, grows more and more dull, until the limit of absolute cardiac dulness is reached. The reason of this change in the note will be fully discussed when the subject of percussion comes to be treated of as a whole in a subsequent chapter.

It is by noting the point at which this change in the note occurs that the position of the outer margin of the heart can be determined by percussion. This is called the area of *relative cardiac dulness* (see fig. 15). Now, although it is in most cases not difficult to sketch out the entire area of relative dulness, yet for ordinary clinical purposes it is only necessary to



percuss in two directions—vertically, parallel to the left margin of the sternum, and transversely, at the level of the fourth rib.

(1.) *The verticle line* is thrown about one inch to the left of the sternum in order to avoid the aorta. In this direction the full note of the lung becomes impaired at about the lower edge

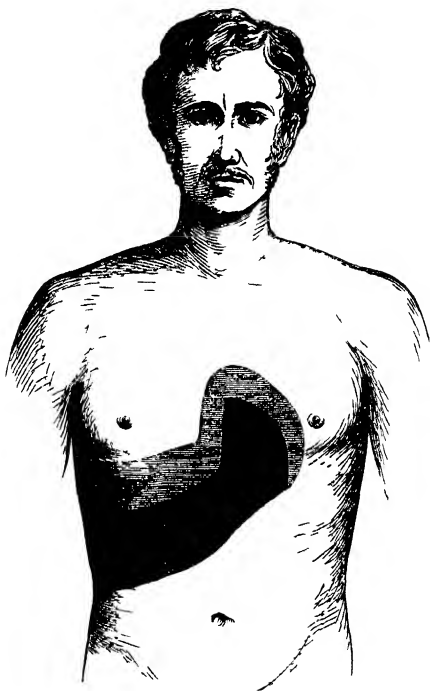


FIG. 15 —Cardiac and Hepatic Dulness.

- |                            |                             |
|----------------------------|-----------------------------|
| 1 Relative hepatic dulness | 3 Relative cardiac dulness  |
| 2 Absolute hepatic dulness | 4. Absolute cardiac dulness |

of the third rib. This is, then, the limit of relative cardiac dulness in this situation. Prolongation of dulness upwards above the third rib (if it be not caused by dislocation of the heart upwards, by pulmonary consolidation, or by the bulging

of an aortic aneurism) arises from pericardial effusion. On the other hand, increase of dulness at the *lower* end of this parasternal line (if the heart be not dislocated downwards) indicates enlargement of the left lobe of the liver.

(2.) *The transverse line* at the level of the fourth rib. The *left border* of the heart is usually marked with sufficient accuracy by the position of the apex-beat, but in cases of uncertainty it may be determined by percussing from without inwards in the direction indicated, when it will be found that the margin of relative dulness lies a short distance ( $\frac{1}{2}$  to 1 inch) to the left of the absolute cardiac dulness. Increase of the dulness to the left usually indicates hypertrophy and dilatation of the left ventricle. The *right margin* of relative dulness at the level of the fourth rib indicates the amount of dilatation of the right auricle. It is usually found about one inch to the right of the margin of the sternum. Increase of dulness in this direction, therefore, takes place when the right auricle is distended, and indicates obstruction to the circulation. This obstruction is probably aortic, if the apex-beat is displaced downwards and outwards. If this be not the case, it is either mitral or pulmonary.

In measuring transversely the extent of the cardiac dulness, it is necessary to bear in mind the curve of the thorax.

The aortic dulness exists in the normal condition simply as a slight rounded projection from the upper part of the relative cardiac dulness. It rises as high as the upper margins of the second costal cartilages.

When dilatation of the aortic arch takes place, the relative dulness becomes better marked, and passes upwards and towards the right, impairing the note over the *manubrium sterni*. In aneurism of the aortic arch, the area of dulness increases correspondingly, and if the tumour approach near to the sternum there is produced an area of absolute dulness.

When the aneurism comes to press firmly on the breast-bone, the dulness which this gives rise to is not absolutely limited to the site of the tumour, but extends up and down the sternum to a variable distance. This dulness is probably in some measure

caused by the obstruction to the vibration of the sternum which the firm pressure of the tumour presents. A similar alteration in the percussion note may be artificially produced by pressing heavily on the sternum with the hand while percussion is being made at a neighbouring point.

## CHAPTER XI.

### Circulatory System—(continued).

#### AUSCULTATION OF THE HEART.

CARDIAC auscultation is almost invariably practised with the aid of a stethoscope. For ordinary purposes, a simple wooden instrument suffices, the cup of which accurately suits the ear of the auscultator; but in exceptional cases a double stethoscope, such as that of Alison, may with advantage be used, which fits into both ears.

On listening over the heart, two sounds are to be heard, separated by two pauses of unequal length. The first sound, which is considerably the more prolonged of the two, is followed by a short pause: to it succeeds the short second sound, and finally a long pause. At the apex of the heart the first sound is the louder of the two, the rhythm being there trochaic (— ∪), while at the base the accent is thrown upon the second sound, as in the iambus (∪ —).

Associated more or less intimately as the sounds are with the valves of the heart, it is necessary for the observer to have a clear conception of the position which these structures occupy in relation to the anterior thoracic wall.

*The Mitral Valve*, which is situated on a plane considerably posterior to those in which the other valves lie, may be said to correspond to the sternal end of the third left costal cartilage, projecting more or less upwards and downwards into the adjacent intercostal spaces.

*The Tricuspid Valve.*—The attached edge of this valve corresponds to a line drawn slantingly across the sternum from the third left intercostal space to the fifth right costal cartilage.

*The Aortic Valves* lie horizontally, opposite a line joining the middle of the sternum and the inner end of the third left costal cartilage.

*The Pulmonary Valves* are also placed horizontally, slightly higher and more to the left than the aortic, corresponding to the upper border of the third left costal cartilage, or to the second left interspace.

It will thus be seen that, in relation to the chest-wall, these valves lie very close to one another, a superficial area of half-an-inch square including a portion of all four (Walshe). The sounds produced in connection with these valves, are, however, best heard, not immediately over them, but at that point on the chest-wall at which the cavity into which the vibrating blood is flowing approaches nearest to the surface. Naturally this point where the sound is most intense varies in each case, and hence we have four areas for auscultation.

The *Mitral Area* is a circle about an inch in diameter, surrounding the apex-beat. This is the only point at which the left ventricle comes in contact with the chest-wall.

The *Tricuspid Area* embraces the lower part of the sternum, particularly the left border at the level of the fourth, fifth, and sixth cartilages.

The *Aortic Area.*—The aorta approaches nearest to the chest-wall at the second right costal cartilage, and consequently the aortic area is situated at this point.

The *Pulmonary Area* corresponds to the second left intercostal space (Von Dusch), or to the third left costal cartilage.

The causes of the heart sounds heard in these various areas may be briefly indicated as follows:—

The *first sound* (systolic), heard in the mitral and tricuspid areas, and synchronous with the ventricular systole, is formed partly in connection with the left ventricle, and partly in connection with the right. In each case its mode of production is

similar, and depends (in all probability) upon two factors. The first of these, as was shown by Rouanet as early as 1832, is the vibration of the auriculo-ventricular valves, arising from the sudden tension caused by the ventricular contraction; and the second is the muscular vibration in the muscular fibre of the ventricle, which takes place during its contraction (muscle sound).

The first sound, as heard in the aortic and pulmonary areas, is probably to be considered as due to the conduction of the mitral and tricuspid sounds to the base of the heart.\*

The *second sound* (diastolic), heard in the aortic and pulmonary areas, has its origin in the vibrations produced in the semilunar valves at the orifices of each of these vessels during the diastole of the heart, by the blood being forced back upon these valves.

The second sound, as heard in the mitral and tricuspid areas, is merely the aortic and pulmonary sound conducted to the apex.

It is evident that we have thus to deal with four sounds during the course of the cardiac revolution, which arise entirely independently of one another.

Two systolic sounds originate at the mitral and tricuspid valves, and in the muscular fibre of the ventricles; and two diastolic sounds are caused by vibration of the semilunar valves at the aortic and pulmonary orifices.

Of these, the first two are synchronous, and are consequently heard as one sound, and, as the last two also take place almost simultaneously, only two sounds, a systolic and a diastolic, are audible over the cardiac area. The systolic sound marks accurately the commencement of the ventricular systole, and the diastolic expresses with equal precision the instant at which the diastole of the heart begins.

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\* According to Heynsius, however, the first sound, at the base of the heart, arises independently at the orifices of the aorta and pulmonary artery, and is the result of vibrations arising in the blood currents which are propelled into these two vessels during the ventricular systole.

The changes in the cardiac sounds which disease produces are of two varieties—(1) Alterations in the normal heart sounds, and (2) murmurs, or adventitious and abnormal sounds.

#### MODIFICATIONS OF THE NORMAL HEART SOUNDS.

The normal sounds may be modified in disease as regards intensity, purity, quality, &c. It will suffice to consider such changes under the three following heads:—

##### 1. **Variations in Intensity** (intensified or enfeebled)—

###### (a.) *Intensified.*

This takes place with regard to all the heart sounds under the influence of mental or bodily excitement, pyrexia, &c., or may be the result of improved conduction, either because the chest walls have become thin, or from condensation of the lung tissue conducting the vibrations more distinctly to the surface.

When the accentuation is limited to one sound, as heard in a particular area, it may result (1) from the better conduction through consolidated lung tissue (particularly in the case of the second sound at the base of the heart), (2) from hypertrophy of the walls of a particular cavity of the heart, or (3) it may arise from increased tension in the column of blood which presses back on the valves.

Intensification is most significant when found in connection with the second sound in the aortic or pulmonary areas. In order to determine that either of these sounds is accentuated, it is only necessary to compare it with the other, always bearing in mind, however, that in the normal condition the aortic sound is louder than the pulmonary.

*Accentuation of the aortic second sound* arises from increase of the arterial tension. It is also met with in dilatation of the aortic arch, and is well marked in cases of aneurism of the arch, where there is hypertrophy of the left ventricle, but in which the sigmoid valves are still competent.

*Accentuation of the pulmonary second sound* is found wherever there is increased pressure in the pulmonary artery. It is a sign that some hindrance exists to the circulation in the lungs, and it is well marked in cases of mitral stenosis and mitral incompetence.

(b.) *The sounds may be enfeebled* by reason of bad conduction through thick chest-walls, emphysematous lungs, pleural effusions, &c., or they may be audible with difficulty on account of loud sounds in the neighbouring lungs. Weakness of particular sounds is chiefly seen in connection with affections of the myocardium—as after typhus—in fatty degeneration of the cardiac muscles, &c.

**2. Impurity of the Sounds.**—A heart sound is said to be impure when it wants the clearness and definition of normal sounds, or when it consists of or is accompanied by irregular vibrations. Such slight changes do not amount to a murmur, but in practice an impure sound is not readily distinguished from one accompanied by a soft murmur.

Impurity in the heart sounds may be caused by thickening of the valves, irregular tension, and closure of the different cusps of the valve. It is of little diagnostic importance.

**3. Reduplication of the Heart Sounds.**—Not uncommonly the heart sounds become doubled, each cardiac cycle giving rise to three, or even four, separate sounds. On careful examination, it will be found that one or other sound has become broken up into two. Reduplication frequently occurs in health, and is then intimately associated with respiration and the changes in the intra-thoracic pressure thereby produced. The first sound is reduplicated at the end of expiration and the beginning of inspiration, while the second sound is doubled at the end of inspiration and the beginning of expiration. Such reduplication has no peculiar significance, and only indicates that, in the case of the first sound, the auricular pressure on the right side of the heart is increased, and retards the closure of the tricuspid valve;



and in the case of the second sound, that the pressure in the pulmonary artery is increased, and so the closure of the pulmonary valve is accelerated.

In disease, reduplication is more marked and constant. It is frequently found in connection with the second sound in cases of mitral constriction and lung disease, from the abnormally high tension in the pulmonary circulation thereby produced. It may also arise in cases of aortic stenosis. Reduplication of the first sound may be produced by irregular contraction of papillary muscles, or, in very rare cases, by asynchronous contraction of the ventricles, in addition to its origination from differences of blood tension, as in health.

#### MURMURS.

The murmurs which are met with in connection with the heart's action are divided into two groups—(1) *Endocardial*, or those which arise within the heart; and (2) *Exocardial*, or those originating in connection with the outer surface of that organ.

**1. Endocardial Murmurs.**—Murmurs, or abnormal sounds, differ from the natural heart sounds in being more prolonged, and less sharply defined. Those which are of endocardial origin all arise from oscillations or vibrations in the blood stream as it passes through a narrow opening into a wider space beyond.

To go more particularly into the physical question of the origin of murmurs, we must put away from our minds the idea that such murmurs are ever caused by rubbing of the blood stream upon roughnesses or irregularities on the valves or orifices of the heart. Such a state of matters is physically impossible; for when a fluid streams through a tube, *the walls of which it wets* (as the blood does the endocardium), a thin layer of the fluid becomes attached to the inner wall of the tube by the force of cohesion, and consequently seeing that the current itself never comes in contact with the tube-wall, no friction between the two is possible (Neumann, Helmholtz).

When a fluid is passing along a tube of uniform calibre, no murmur arises, unless the rapidity of flow is very great. The blood current is never rapid enough to give rise to a murmur under these conditions. But when a constriction exists in the tube, and the fluid is thus forced to pass from a narrow into a wider portion, a murmur readily arises; and the greater the difference between the lumen at the two points, the more easily is the murmur produced; or, in other words, the less rapidity of current is required for its production.\* It is in this way, and under such conditions, that all such cardiac murmurs arise. Whenever the blood stream passes with sufficient velocity through a narrow orifice into a wider space beyond, there will be such friction between the fluid particles as to give rise to sonorous vibrations in the fluid, as when, for example, a rent occurs in the aortic or mitral valves, or when the orifices they guard become narrowed by disease.

In the production of murmurs there enters, however, another factor which must not be entirely lost sight of—viz., the condition of the blood. So early as 1837, some observations were made which seemed to show† that the ease with which murmurs could be produced by driving fluids of different density through veins stood in inverse ratio to the density of the fluid, and this conclusion has been arrived at by many subsequent experimenters, among whom may be mentioned Weber,‡ Nolet,§ and Richardson.|| It was found, in particular, that when water was added to blood a murmur was more easily produced than when blood alone was used, and this observation has a very

\* That such murmurs are not due to the vibration of Savarts' "veine fluide" is shown by the fact that the murmur may arise in tubes of uniform calibre where no such "veins" are formed.

† *Vide* Williams, on the "Pathology of Diseases of the Chest." London, 1840.

‡ *Arch. f. physiol. Heilkunde*, xiv.

§ *Arch. der Heilkunde*, xii. 1871.

|| *Med. Times and Gazette*. 1868.

important bearing upon anæmic murmurs, and particularly those which arise in veins, as will be hereafter shown.\*

*Endocardial Murmurs* are of two varieties—(1) Those of valvular origin, and (2) those of other than valvular origin.

Of these, the latter class is so rare that it is well invariably to endeavour to associate a murmur with a particular valve or orifice, and only in the event of failure in such an attempt, to consider the possibility of a non-valvular cause.

Having ascertained the presence of a murmur, there are three points which should be carefully noted—(1) The rhythm of the murmur—i.e., the particular period in the cardiac contraction with which it corresponds (auricular or ventricular systole or diastole, &c.); (2) the point of maximum intensity and the direction of propagation; (3) the condition of the normal heart sound at the valve or orifice at which the murmur is supposed to originate.

(1.) *Rhythm*.—To ascertain the rhythm of a murmur it is necessary to lay a finger upon the apex-beat or upon the carotid artery while we auscultate. This gives the time of the ventricular systole, and enables us to say which is the first and which the second sound, and consequently the rhythm of the murmur can be readily ascertained. If, however, the cardiac pulsations exceed 90 per minute, it may be impossible thus to time the murmur, and in such cases we must wait till rest and appropriate medication have reduced the rapidity of the action of the heart.

(2.) *Point of Maximum Intensity and Direction of Propagation*.—It has been already stated that the normal heart-sounds are heard with most distinctness in various areas according to the valve or orifice at which they arise. These sounds are

\* I think it likely however, that it will ultimately appear that the density of the fluid is not the most important physical condition in determining the production of murmurs. Viscosity will probably be found to possess much greater importance in this regard. In the absence of exact experiments, it is impossible to be certain on these points.

conducted in the direction of the blood current, and are best heard where the cavity into which the current flows approaches nearest to the surface of the body. The same holds good for murmurs, every endocardial murmur of valvular origin having its points of maximum distinctness in one of these four areas, and being of mitral, tricuspid, aortic, or pulmonary origin, according as it is best heard in the mitral, tricuspid, aortic, or pulmonary area. Two exceptions to this rule however exist—viz., (1) a mitral systolic murmur, which is sometimes best heard an inch to the left of the pulmonary area, and (2) an aortic diastolic murmur, which is occasionally most intense at the xiphoid cartilage.

Having satisfied ourselves as to the rhythm, and the point of greatest intensity (and consequently the seat of origin) of the murmur, it is a matter of simple reasoning to discover its mode of production. Thus for example, a systolic mitral murmur can only be one of regurgitation through incompetence of the valve. A præsystolic mitral murmur, on the other hand, must result from stenosis of the mitral orifice, since it occurs at the instant when the blood is being propelled by the auricular systole through the mitral orifice into the ventricle. We shall consider the causation of each particular murmur further on.

The direction of the conduction of murmurs may be of use in indicating their origin. Stated generally, it may be said that systolic mitral murmurs are conducted towards the left axilla, and to the angle of the left scapula; tricuspid murmurs are heard over an area corresponding to the right ventricle; aortic murmurs are propagated up and down the sternum and into the arteries; and finally, pulmonary murmurs are usually not audible outside of that area.

(3.) *The Condition of the Normal Sound at the Orifice at which the Murmur originates.*—The presence of a normal sound, more or less obscured by the accompanying murmur, indicates that the valve is not entirely destroyed. The method of auscultation suggested by Gendrin is of value for the purpose of ascertaining

this. He recommends the ear to be slightly raised from the stethoscope, the instrument remaining unmoved, when the sound will become more and the murmur less audible. The real value of the presence of a cardiac sound as an indication of the state of the valve is very questionable. In the case of aortic disease, the auscultation of the arteries gives much more reliable results.

The character of the murmur (soft, blowing, rasping, whistling, &c.) is of little diagnostic value. As a general rule *direct* murmurs (those which arise in the blood current as it is flowing in its normal direction) are rough; whereas *indirect* murmurs (those which flow in a direction contrary to that of the normal current) are soft.

Having determined the rhythm and seat of the murmur, it is, as I have already said, no very difficult matter to infer the manner of its causation. This is done by simply bringing to mind what is happening at the valve in question during the particular period at which the murmur is heard. In order to make this plain, we will now consider very briefly the various murmurs met with in connection with each valve.

#### (a.) *Mitral Murmurs.*

Mitral murmurs are systolic, diastolic, or præ systolic in rhythm, according as they occur during the ventricular systole, the diastole, or immediately before the ventricular systole—i.e., during the auricular systole.

*Mitral systolic murmurs.*—These murmurs, originating at the mitral valve during the ventricular systole, indicate that from some cause the valve does not completely cover the orifice, but allows a part of the blood contained in the ventricle to be forced back into the auricle. As a result of this, the blood-pressure in the auricle rises and its cavity becomes dilated, and when this stretching has reached a certain point, the backward pressure is transferred to the pulmonary veins, to the capillaries of the lung, and thence to the pulmonary artery. The increased

resistance in that vessel causes the right ventricle to dilate, and subsequently to hypertrophy. The necessary result of this hypertrophy of the right ventricle is that the second sound in the pulmonary area becomes accentuated, for the blood-pressure in the pulmonary artery is raised, and consequently the rebound of the blood column upon the pulmonary valves after the ventricular systole is rendered more forcible. There are thus three chief physical signs to be looked for in cases of mitral incompetence—(1) the systolic murmur, (2) the hypertrophy or dilatation of the right ventricle, and (3) accentuation of the pulmonary second sound. To these there came to be added, subsequently, hypertrophy of the left ventricle, dilatation and hypertrophy of the right auricle, irregularities of rhythm, and other signs.

This incompetence of the mitral valve is the result of one of two processes; either the valve itself has become altered in shape, or the orifice has increased in size, so that the valve which formerly sufficed to occlude it is not now sufficiently large. The former condition arises as a result of sub-acute and chronic endocarditis in connection with rheumatism, syphilis, scarlatina, &c. The valves become thickened and shrivelled up, and so the incompetence originates.

The second condition (*viz.*, increase in the size of the mitral orifice) is found in all diseases in which relaxation of the cardiac muscle takes place to a marked extent, as in typhus, typhoid, relapsing, and rheumatic fevers; in scarlatina, measles, erysipelas, small-pox; and not least importantly, in chorea and in various forms of anæmia, particularly in chlorosis. In all these conditions, the myocardium is so softened and stretched as to allow the mitral orifice to become too large to be covered by the valve. Regurgitation of blood takes place into the auricle; and in passing from a narrow orifice into a cavity, sonorous vibrations arise in the fluid, and in these cases the formation of a murmur is no doubt aided by the watery condition of the blood, in the manner already described (page 117).

Mitral systolic murmurs are usually heard with greatest

distinctness in the mitral area, and are propagated towards the left axilla and the angle of the left scapula. Occasionally, however, they are most audible a little to the outside of the pulmonary area, especially in the case of anæmic murmurs; and this probably arises (as Naunyn has pointed out) because the sonorous vibrations in the auricle are conducted into the auricular appendix, and become most audible at that point on the chest-wall where the appendix approaches nearest to the surface—*i.e.*, about an inch and a-half to the left of the pulmonary area.

*Mitral præ systolic and diastolic murmurs* arise from the same cause—*viz.*, stenosis (narrowing) of the mitral orifice. Immediately after the ventricles of the heart have contracted they relax and begin to refill with blood, and during the period of time represented by the second, or diastolic sound, and by the long pause, this process of filling goes on. At first the blood follows the retreating walls of the ventricles, propelled partly by gravity and partly by the ordinary intra-thoracic pressure, and so flows slowly through the patent orifices (mitral and tricuspid) into the respective ventricular cavities. But towards the end of the long pause, the auricular contraction takes place, and the remainder of the blood is thus more powerfully forced into the ventricles. In ordinary circumstances these actions take place noiselessly; but when stenosis of the mitral orifices arises (we speak now of the left side of the heart alone) as a result of endocarditis, the narrowing may be sufficient to throw the fluid into sonorous vibrations. It depends on the rapidity of flow, and the narrowness of the orifice in relation to the size of the ventricular cavity, whether or not a murmur will occur,—and if so, whether it will be diastolic or præ systolic in rhythm, or, in other words, whether it will be produced when the blood is flowing into the ventricle immediately after the ventricular systole, or later on, during the auricular systole.

These murmurs sometimes co-exist, and may either run into one another, and so fill up the whole time occupied by the ventricular diastole, or they may be separated by a very short

interval of silence. The diastolic portion is usually soft, whilst præ systolic (or auricular-systolic) murmurs are almost invariably rough in character.

Mitral stenosis is followed by much the same physical signs as have been mentioned in connection with mitral incompetence. The right side of the heart hypertrophies and dilates, and the left auricle undergoes similar changes. There is a peculiar thumping character about the first sound, accentuation and often reduplication of the second pulmonary sound, and usually a well-marked præ systolic thrill at the apex. The rhythm of the cardiac pulsations is almost always irregular.

#### (b.) *Tricuspid Murmurs.*

Tricuspid murmurs resemble those at the mitral valve in regard to their causation.

*Systolic tricuspid murmurs* are indicative of incompetence of the valve, with consequent regurgitation of blood into the right auricle during the ventricular systole. This results either from deformity of the valve, produced, as in the case of the mitral valve, by endocarditis, or from dilatation of the orifice. The latter condition may be occasioned by such causes as produce a corresponding state of matters on the left side of the heart (fevers, anæmia, &c.), but more commonly this relative incompetence, as it has been called, is caused by distension of the right auricle and ventricle, the result of obstruction to the circulation through the lungs, produced most distinctly in cases of stenosis or incompetence of the mitral valve. Tricuspid regurgitation should not be diagnosed, unless the signs in connection with the jugular veins formerly described are present.

*Præ systolic tricuspid murmurs* are very rarely met with, and never without other valvular complications. They are the result of stenosis of the tricuspid orifice, and the mechanism of their production is similar to that which produces the corresponding mitral murmur.



(c.) *Aortic Murmurs.*

Aortic murmurs are of two varieties—systolic and diastolic. These usually coexist.

*Systolic aortic murmurs* are those produced at the aortic orifice as the blood is propelled into the aorta by the contraction of the left ventricle. Such a murmur arises when the orifice is contracted or roughened as a result of endocarditis. The murmur is usually loud and sawing, occasionally musical, and whilst it is loudest in the aortic area, it can most frequently be heard over the whole front of the heart.

Aortic stenosis leads first to dilatation, and second to hypertrophy of the left ventricle, owing to the extra resistance which the ventricle has to overcome in forcing the blood through the aortic orifice. This hypertrophy, and the small hard pulse which will be mentioned presently, are the most important signs to which aortic obstruction gives rise, in addition to the systolic murmur, the character and localisation of which have just been described.

Aortic incompetence leads to even more marked hypertrophy of the left ventricle than obstruction does. The very peculiar pulse which is found in such cases (*Pulsus celer*, Corrigan's pulse) is of considerable importance in diagnosis, and will be described hereafter.

*Diastolic aortic murmurs* are the result of incompetence of the aortic valves, the blood regurgitating from the aorta into the left ventricle during the ventricular diastole. The position of maximum intensity of this murmur varies very much. In many cases it is best heard in the aortic area; not uncommonly it is loudest at the ensiform cartilage; rarely, the apex-beat is the situation at which it is most distinct.

Most usually these two murmurs are heard together, the so-called double aortic murmur, for the valves are rarely incompetent without presenting some obstruction to the flow of blood over them into the aorta.

*(d.) Pulmonary Murmurs.*

Among these pulmonary murmurs we do not include those hæmic murmurs which arise at the mitral valve, and have their seat of greatest intensity an inch or more to the left of the pulmonary area.

True pulmonary murmurs are of very rare occurrence. They are systolic and diastolic in rhythm.

*Systolic pulmonary murmurs* are either inorganic or organic. The former have been supposed by Quinke to be produced where from some cause the left lung is retracted, and the heart in its systole so compresses the pulmonary artery as to give rise to sonorous waves in that vessel.

Organic systolic murmurs are almost invariably due to congenital constriction of the pulmonary artery. Such cases are rare, and differ much from one another according to the period of cardiac development at which the constriction commenced. The ventricular septum is usually deficient, with cyanosis as a consequence.

*Diastolic pulmonary murmurs* are still more rare. They result from incompetence of the pulmonary valves, and are invariably accompanied by systolic pulmonary murmurs.

*Endocardial murmurs of non-valvular origin* are probably of very rare occurrence indeed. They may result from—

(1.) Congenital deficiency of some part of the septum, which divides the two sides of the heart; and in that case they only intensify the valvular murmurs already existing.

(2.) Flakes of lymph attached to the valves are said to cause such murmurs.

(3.) Changes in the density of the blood in anæmia, chlorosis, &c., may allow of murmurs forming under conditions under which no such sonorous vibrations would arise in blood of normal composition. It has been already pointed out that many of the hæmic murmurs are mitral in their origin, resulting

from incompetence caused by relaxation of the cardiac muscle. A small proportion of these murmurs may, however, arise in the blood-stream, where no incompetence exists. Such murmurs are soft, invariably systolic, and are usually heard most distinctly over the base of the heart.

**2. Exocardial Murmurs.**—These murmurs are caused by the friction of the two pericardial surfaces on one another, when the surfaces have become roughened as a result of pericarditis, &c. Such friction murmurs are, for the most part, readily distinguished from endocardial murmurs. They are rough and grating, never blowing. They are localised, and are not propagated in the direction of the blood current; and as they usually arise first towards the middle of the heart, the point of greatest intensity does not generally coincide with any one of the cardiac areas. They can always be perceived by the hand, if at all intense, which is only exceptionally the case as regards endocardial murmurs. Further, the rhythm of exocardial murmurs is irregular. They are not confined to any particular phase of the cardiac action, are neither permanently systolic nor diastolic, but vary from minute to minute.

Exocardial murmurs are also sometimes occasioned by friction of two roughened surfaces of the pleura overlying the heart on one another. Such friction murmurs vary in intensity with the movements of respiration.

## CHAPTER XII.

### **Circulatory System—***(continued).*

#### THE EXAMINATION OF THE ARTERIES, CAPILLARIES AND VEINS.

##### ARTERIES.

THE physical examination of the arteries may be conducted by means of inspection, palpation, percussion, and auscultation. Of these we will speak in their turn.

**Inspection.**—In health, the pulsation of the arteries of the body is but little visible, except under the influence of mental emotion or bodily strain. As the result of disease, however, pulsation may become visible in all the superficial arteries of the body, particularly in the carotid, temporal, and radial vessels. All disturbances of cardiac innervation, such as arise in Graves' Disease, and all feverish conditions, are liable to produce such excited action of the heart as will occasion this visible pulsation. Still more marked is the pulsation when the left ventricle is hypertrophied, and, above all, when the aortic valves have been rendered incompetent. Dilated tortuous, and visibly pulsating temporal or radial arteries are usually found to have undergone atheromatous changes; and finally, inspection may show us the localised pulsation of an aneurism.

**Palpation** of the arterial system is almost confined to the

radial artery,—the carotid, brachial, and femoral being but rarely palpated.

*The radial pulse* is, in health, equal on the two sides; but abnormal distribution, compression, or other pathological condition may so act as to make one pulse weaker than the other. So also, the pulse-wave propagated from the heart outwards towards the periphery may not arrive at the two wrists synchronously. This condition occurs where there is simple or aneurismal dilatation of the aortic arch, and is particularly noticeable if the aneurism be situate between the innominate and the left subclavian. It is to be observed that the interval of time which occurs between the cardiac systole, and the arrival of the blood-wave at the wrist may sometimes be considerably longer than usual. Such delay arises either from stenosis of the aortic orifice rendering the systole slow and difficult, or from aortic incompetence where (as Tripier has pointed out with great probability) the onward wave meets with, and is delayed by, the regurgitating blood.

It may probably conduce to greatest clearness if the conditions of the pulse are considered under three headings—viz., (1) frequency, (2) rhythm, (3) character.

(1.) *Frequency of the pulse*, which in the male adult averages about seventy beats per minute (slightly higher in women), varies in healthy individuals according to the age, according to the time of day and the external temperature, and may be greatly influenced by mental emotions, and by the administration of certain drugs. In disease the pulse is sometimes abnormally slow; as for example, in jaundice, in fatty degeneration of the heart, and in some affections of the brain. More frequently, however, the pulse rate is increased in rapidity. The rapid pulse of fever, of collapse, and of the various cardiac neuroses is well known. Very generally the pulse is rapid in diseases of the valves of the heart, particularly the mitral.

2. *Rhythm*.—The radial pulsations, which are normally separated by regular intervals of time, and so are rhythmical, may be altered in this relation to each other in a great variety

of ways, the normal rhythm being sometimes changed into total irregularity, while at other times the beats, although following each other in an abnormal manner, still possess a certain rhythm. Amongst the latter may be mentioned the (1) *pulsus bigeminus*, in which each two beats form a group separated from the two which precede and the two which succeed by longer pauses than the interval which separates each pair; (2) the *pulsus paradoxus* is that variety of pulse, so carefully described by Kussmaul, where with each inspiration the pulse wave becomes smaller, or is completely lost. When it is present in all the arteries of the body, it may be due to one of two causes—either to fibrous adhesions between the aorta and the sternum, or to some other obstruction which during inspiration prevents the free passage of the blood into the aorta; or it may result from any obstruction to the entrance of air into the lungs, which during inspiration lessens the pressure within the thorax. When the *pulsus paradoxus* occurs only in one radial artery, it is due, as Weil has pointed out, to inflammatory adhesion between the pleura and the subclavian artery. In the *pulsus alternans* there is a regular alternation between a small and a large pulsation. When, after a series of regular pulsations, one or more beats are omitted, the pulse is said to be *intermittent*. The intermissions, due either to momentary cessation of the heart's action or to the blood-wave in question being too feeble to reach the wrist, may be regular or irregular, and often occur independently of heart disease. Most frequently, however, the intermittent pulse is associated with some cardiac affection, generally mitral disease. Very irregular pulsations, in which no rhythm of any kind can be detected, are commonly (although by no means always) due to affections of the mitral valve, generally to mitral constriction, of which affection an extremely irregular pulse, even in the early stages, is an important symptom, and one to which considerable diagnostic importance may attach.

3. *The character of the pulse* varies in a great number of ways, giving rise, especially in the works of the older writers,

to a very extensive nomenclature. It will be sufficient for ordinary purposes to notice the following points:—

(a.) The *expansion* of the pulse. A pulse which reaches its full expansion quickly, and as rapidly collapses again, giving to the finger the impression of a very quick stroke, is denominated the *pulsus celer*, and this celerity is, as Corrigan first pointed out, most distinct where there is aortic incompetence (hence called Corrigan's pulse). The opposite condition, the *pulsus tardus*, is distinguished by the slow manner in which the artery fills and empties, and this sluggishness may be due to slowness in the contractions of the heart, to a hindrance in the capillary and venous circulation, or to loss of elasticity in the arterial wall itself. It is perhaps most frequently met with as a result of arterial sclerosis.

(b.) The *tension* of the pulse, or, in other words, the blood-pressure on the inner surface of the artery, may be approximately estimated by the pressure of the finger required to obliterate the pulse.\* When the tension is high (as in hypertrophy of the left ventricle, lead colic, peritonitis, &c.), we speak of a *hard* or *tense* pulse, and under the reversed circumstances (as in mitral disease), of a soft and compressible pulse. Above all things, however, it must be borne in mind that the impression of tension or hardness may be given to the finger by a rigid condition of the arterial wall, and it is only when this factor can be eliminated that any safe deductions can be drawn regarding the blood pressure itself. When the radial artery has undergone calcification, the irregular prominences can usually be felt, and this will prevent error.

(c.) The *volume* of the pulse. A full pulse may be produced by one or more of three factors, powerful ventricular contraction, loss of elasticity of the arterial wall, and interference with the blood flow from the arteries into the capillaries. The opposite conditions may give rise to an empty pulse. The pulse is also spoken of as large or small, tremulous, thready, &c.

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\* The tension may be more accurately estimated by means of the sphygmomanometer of Von Basch, which will be hereafter described.

All these varieties of pulse are best studied with aid of the sphygmograph.

**Percussion of the Arteries** is almost entirely limited to cases of thoracic aneurism, of which mention has been already made.

### **Auscultation of the Arteries.**

1. *In health.*—As in cardiac auscultation, so also in auscultation of the arteries, we have to distinguish two phenomena—sounds and murmurs. In health, if the stethoscope be placed over the carotid artery as lightly as possible, two sounds are usually to be heard, corresponding respectively to the expansion and contraction of the artery. Of these the latter is simply the second aortic sound conducted into the carotid, and it seems most probable (Weil, Heynsius\*) that the sound coinciding with the arterial expansion ought also to be regarded as the conducted aortic systolic sound (Guttman,† however, regards it as in part originating in vibrations of the arterial wall). These two sounds can also generally be heard in the subclavian, and occasionally the first can also be detected in the abdominal aorta and in the brachial and femoral arteries; but in the more peripheral vessels no auscultatory phenomenon is present in health. If pressure be made with the stethoscope upon an artery, such as the brachial just above the elbow, where normally no sound can be heard, the narrowing of the lumen of the vessel thereby occasioned gives rise to vibrations in the blood stream, and to an audible murmur coincident with the arterial expansion. If the pressure be increased, this murmur passes into a sharp sound.

2. *In Disease.*—Sounds or murmurs may be heard in the arteries under three pathological conditions—

(a.) *Murmurs conducted from the Heart.*—It is, as a rule, aortic murmurs (both systolic and diastolic) which are propagated into the arteries, although mitral murmurs are occasionally to be heard very faintly in the carotids.

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\* *Loc. cit.*

† “*Lehrb. der Untersuchungs Methode.*”



(b.) *Sounds and Murmurs originating in the Arteries in consequence of general Circulatory Disease.*—In aortic incompetence a sound coinciding with the arterial expansion may be heard in all the accessible arteries of the body, due almost certainly to the rapid transition from extreme relaxation to extreme tension which the arterial coats then undergo. A double sound over the femoral artery is also sometimes to be heard in such cases, as was first pointed out by Conrad\* and subsequently more fully studied by Duroziez,† Traube,‡ Friedreich,§ and others, and lately by Senator.|| The first of the sounds, that coinciding with the arterial expansion, originates in the arterial coats, as already described; and the second arises, in the majority of cases, not in the artery, but in the femoral vein, as a result of coexisting tricuspid incompetence. Very rarely, indeed, cases occur in which the latter sound is of arterial origin (the tricuspid valve being intact), and it then results from aortic incompetence.

A double murmur may be produced in the femoral artery in cases of aortic incompetence by pressure with the stethoscope, the one murmur being caused by the pulse wave, the other by the returning backward wave, which in such cases flows towards the heart during the arterial collapse. This double murmur may also occasionally be heard in cases of anæmia, typhoid fever, &c.

(c.) *Murmurs originating in the Arteries in consequence of Local Changes.*—Such murmurs are to be heard over aneurisms and vascular tumours, but more important are the subclavian murmurs. While occasionally occurring in healthy persons, murmurs over the subclavian arteries are much more frequently heard in cases of phthisis, due probably to adhesions between the pleura and the vascular walls, and hence much influenced by the respiratory movements.

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\* "Zur Lehre über die Auskultation der Gefässe." Giessen, 1860.

† *Arch. gén. de méd.*, 1861.

‡ *Vide Berl. kl. Woch.*, 1867, No. 44.

§ *Deutsches Arch. für kl. Med.*, xxi. p. 205, and xxix., 1881.

|| *Zeitschr. für kl. Med.*, iii., 1881.

The encephalic murmur which Fisher discovered in children has, so far as our present knowledge goes, no diagnostic significance.

The cephalic murmur which Tripier has recently shown to be present in the eyeball in anæmia, is supposed by him to be of arterial origin. It is coincident with the expansion of the arteries. Gibson has shown that it is also to be heard over the mastoid process and the occiput, and as it is a little difficult to see why, if it be an arterial murmur, it should be loudest at these three points, he has referred it to the vibrations in the internal jugular vein produced by the systolic stroke of the neighbouring carotid artery, and conducted into the venous sinus. This ingenious explanation does not, however, seem to me satisfactory. I am inclined to regard this cephalic murmur as venous, but as the product of the systolic augmentation of the venous current which takes place within the skull, and within the eyeball, due to the fact that in each case we are dealing with a closed box, which is practically incapable of expansion. Hence, when a sudden increase of arterial blood takes place in them, when the arteries are distended, an equally sudden out flow must occur through the veins. To the vibrations in this sudden venous current I am inclined to ascribe this very interesting cephalic murmur. The watery condition of the anæmic blood is, of course, the important factor.

#### CAPILLARIES.

The state of the capillary vessels need not be specially noticed here, seeing that the more important points have been elsewhere discussed.

#### VEINS.

Knowledge concerning the condition of the veins may be obtained by inspection and by auscultation. Palpation by the fingers, and percussion, are not fitted materially to aid the physician.

**Inspection.**—By inspecting the veins we ascertain, firstly, their state as to fulness; and secondly, whether the blood contained in them undulates or pulsates.

*Overfilling* of the veins results either from local obstruction, when the vein becomes tense on the distal side, and such of the collateral branches as are not compressed enlarge so as to carry on the circulation—or from interference with the venous circulation generally. Examples of the variety of engorgement arising from local obstruction are to be found in cases of thrombosis of any of the larger venous trunks, or where the pressure of an aneurism or other mediastinal tumour gives rise to overfilling of the veins of the arm. The distension of the cervical veins which arises where the general circulation is interfered with, has already been described.

*Undulation of the Veins of the Neck.*—The pulsations in the cervical veins, which correspond to the movements of the heart, have been already remarked upon. It only remains to mention the undulation which the respiratory movements sometimes produce in the jugular veins. When the cervical veins are overfilled, as a result of pulmonary emphysema or of mitral stenosis, each inspiration diminishes the venous distension, while each expiration increases it, and so the veins show a constant undulation.

**Auscultation.**—Although in cases of tricuspid incompetence systolic sounds are occasionally to be heard over the jugular and femoral veins, the only auscultatory sign which here demands attention is the humming murmur, the so-called *bruit de diable* which is very frequently to be heard in chlorotic females over the bulb or dilatation of the internal jugular vein, and more rarely over the large intrathoracic venous trunks, the superior vena cava, and the innominate veins. Venous murmurs in the former are best heard at the right border of the sternum, from the first right intercostal space to the third costal cartilage. The murmur in the right innominate vein is usually loudest at the sternal end of the first right

costal cartilage, and that in the left over the manubrium sterni. Occasionally a venous hum is to be heard in dilated thyroid veins, and in the subclavians, axillary, brachial, and femoral veins. In venous auscultation, it must be borne in mind that the slightest unnecessary pressure with the stethoscope may develop an artificial murmur.

The *bruit de diable*, as met with in the jugular vein (generally loudest on the right side), is usually of a continuous soft humming character, and occurs very frequently in health. Winterich\* detected it in 80 per cent. of the Bavarian cuirassiers whom he examined. Only when the murmur is strong and loud is it pathological, or can it be taken as evidence of the existence of anæmia; and we may, with Friedreich,† define the pathological venous murmur as limited to those cases in which a thrill is perceived when the finger is applied over the jugular bulb, or in which the murmur is sufficiently loud to be heard when the ear is removed a little way from the stethoscope, or to become apparent to the patient himself, and finally, when a murmur can be perceived over the intrathoracic venous trunks.

These venous murmurs appear to depend for their production upon three factors—1st, upon the rapidity of the blood current; 2nd, upon the change in the calibre of the vein at any particular point (such as occurs in a marked manner at the jugular bulb); and 3rd, upon alteration in the quality of the blood, whether this consists in an actual or only a relative increase of the watery elements.

Usually the jugular humming murmurs are continuous, but they very often vary in intensity, and occasionally are actually intermittent. They are influenced in the following ways:—

1. *Changes in the Posture of the Patient.*—When the head is turned to the opposite side, the murmur becomes much intensified, owing to the compression of the vein by the muscles and fascia. Even when no murmur exists when the head is held

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\* *Deutsche Klinik*, 1850.

† *Deutsch. Arch. f. kl. Med.*, vol. xxix. (1881) p. 263.

straight, a faint bruit may be developed when the head is rotated, especially if firm pressure be made with the stethoscope in addition.

Owing to the acceleration of the blood flow in the veins the murmur is louder when the patient sits or stands than in the recumbent posture.

2. *The Movements of Respiration.*—Sometimes the venous murmur in the jugular is only audible during deep inspiration, and if it be continuous it is almost invariably intensified by that action, in both cases, for this reason—viz., that during inspiration the flow of blood in the vein is accelerated. The same usually holds good with regard to murmurs in the femoral vein, although in rare instances the reverse obtains, and we meet with the remarkable phenomenon of a femoral murmur which is expiratory in rhythm,\* this probably resulting from the increased abdominal pressure which the descent of the diaphragm occasions, and which retards the blood current in the femoral vein.

3. *The Movements of the Heart.*—The anæmic murmur in the jugular vein is sometimes diastolic in rhythm, as was first pointed out by Chauveau,† who ascribed it to the increased blood current in the vein which is the result of the diminution of pressure in the superior vena cava produced during diastole, and which stands closely related to the negative diastolic pressure in the ventricle. While this is no doubt one cause of this diastolic venous hum, it appears to the author extremely probable that the cause suggested by Friedreich is likewise operative—viz., that the pulsations of the aorta compress the superior vena cava during the cardiac systole, thus allowing an uninterrupted flow of venous blood during diastole.

Friedreich, *loc. cit.*

† *Gaz. Méd. de Paris*, 1858

## CHAPTER XIII.

### *Circulatory System—(continued).*

#### GRAPHIC CLINICAL METHODS.

WHEN Chauveau and Marey first introduced to the notice of the profession the sphygmograph and cardiograph, it was hoped that a new and more accurate examination of the heart and circulatory system would soon replace the former methods. This hope has not been realised. There is, indeed, little difficulty in obtaining tracings of the pulse wave and heart beat, and these tracings, moreover, are found to vary greatly in different diseases; but the true meaning of these differences is as yet by no means thoroughly understood. The reason for this lies partly in the fact that the meaning of the normal pulse and heart curve has not yet been explained, in all its details, in a fully satisfactory manner. Still, even now, certain trustworthy facts can be obtained by the use of the recording instruments referred to, and the number of these facts will necessarily increase as the characteristics of the normal pulse wave and heart beat, and the modifications which they may undergo in health, become more fully understood. Moreover, the permanence of the records which may be obtained by the use of such instruments, their value in illustrating the history of individual cases, together with the fact that these instruments give results which are more purely objective than those obtainable by other methods, amply justify a somewhat full description of the manner of using the sphygmograph and cardiograph,

together with some account of the results obtainable by their help.

**Sphygmograph.**—The original instrument of Chauveau and Marey, which, since its introduction, has been repeatedly modified in detail by Marey himself and by others, in its present form (as supplied by Breguet) is the favourite form of instrument employed at the present day. Some have sought to introduce the so-called transmission sphygmograph, but it seems to be generally accepted that the advantages which this form of instrument presents in certain particulars, are more than counterbalanced by very obvious defects. All of these transmission sphygmographs are similar in principle, consisting of two closed Marey tambours—one being influenced through the medium of a button pressing on the radial artery, while the second tambour, joined to its fellow by an india-rubber tube, is arranged to move a recording lever, which writes on the blackened surface of a revolving cylinder. The great advantage presented by this form of sphygmograph is that by its means we can obtain curves of practically indefinite length, more especially if the revolving cylinder move round a spiral spindle. On the other hand, these instruments are all more or less cumbersome and expensive, while the introduction of a long column of air to transmit the form of the pulse wave to the recording lever, introduces many serious possible errors. However, for investigation of certain special points, as, for example, where simultaneous tracings of the pulse, heart, and respiratory movements are desired, the transmission sphygmograph is the only instrument which can be employed. Of these Marey's polygraph\* is probably the most perfect and convenient.

On account of its comparatively small cost and convenient size, it is probable that the original Chauveau and Marey's sphygmo-

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\* *Vide* Marey, "La circulation du sang à l'état physiologique et dans les Maladies." Paris, 1881.

graph, in its most recent modification, will still continue, at least for some time, to be the most commonly used instrument. The principle of its construction is to be found in all text-books of physiology, and need not therefore be dealt with here; and I will confine myself to a description of the typical pulse curve, and the modifications which it undergoes in health and in disease.

*The typical healthy curve*, of which the accompanying tracing (fig. 16) is an example, is usually divided into an ascending and descending portion, either or both of which may present certain secondary undulations. In its most typical form (as in fig. 16) the ascending line (*a* to *b*) rises abruptly at first, and afterwards more slowly, till it reaches its highest point. Then descending more obliquely, it usually presents a more or less well-defined notch or indentation (*c*) before it reaches the principal notch or valley (*d*). This latter notch is best known as the dicrotic notch, and is of great importance corresponding as it does exactly to the closure of the aortic valves. After the dicrotic notch, the curve describes a slight elevation before descending



FIG. 16.—Normal Pulse Curve.\*

ing to its lowest level, in the course of which descent a low wave-like eminence (*f*) is not unfrequently to be discovered. Since the point (*a*) corresponds to the opening of the aortic valves, and the point (*d*) to their closure, the artery is, during the time represented by the interval between these two lines, in free communication with the interior of the ventricle,

\* For this curve and those which follow, as well as for much help and advice in the preparation of this chapter, I am indebted to the kindness of my friend, Professor C. S. Roy, of Cambridge University.



while, during the time of the rest of the curve, the artery is cut off from the heart.\* The point *d*, therefore, forms the most natural division of the pulse wave into its more fundamental parts, the causes which influence its form during the first half being essentially different from those which modify the second half. Keeping this fact in view, and premising that it is almost always easy to find in any curves, of whatever form, the point which corresponds to *d* in our typical curve, I now proceed to describe the modifications of the pulse wave which are to be met with.

And first, with regard to changes in the first half of the curve.

*Anacrotic Pulse.*—The typical pulse curve, of which I have given an example above, is not infrequently called dicrotic, owing to the fact that it presents a fairly well-marked notch in its descending part, although some authors prefer to restrict the term dicrotism of the pulse wave to cases in which that notch is abnormally well marked. In contradistinction to the dicrotic pulse, it is the custom to call those pulse waves in which a more or less well-marked notch occurs in the ascending line as anacrotic. The tracing (fig. 17) annexed shows a



FIG. 17 —Anacrotic Pulse Wave.

fairly typical example of the anacrotic pulse wave. It can be seen that it differs from the dicrotic or normal pulse wave only in the part which lies between the lines *a* and *d*; in other words, in that part of the pulse wave which corresponds to the time when the aortic valve is open. We

would, therefore, expect, *à priori*, that this change in the form of that part of the pulse wave must be due to some difference

\* The whole pulse wave is delayed in its transmission from the commencement of the aorta to the radial artery, but the delay of the different parts of the curve is usually tolerably equal, so that the relative distance between the up-stroke and the dicrotic notch remains the same.

in the relation between the quantity of blood thrown out of the left ventricle and the elastic resistance offered by the aorta and larger arteries. Let us suppose that the arteries are relatively lax, and that the quantity of blood thrown out of the ventricle is not above normal, then it is not difficult to understand that the ventricle will more readily and more rapidly empty itself than when the vessels are relatively rigid. The result of this is that the point of the pulse wave, where the highest pressure exists, and which corresponds to the highest point of the pulse curve, will occur nearer its commencement than would otherwise be the case. Let us, on the other hand, suppose an extreme case, in which the arteries are very rigid, as in well-marked atheroma or calcification of the larger vessels, these latter, as the contents of the ventricle are forced into them during systole, do not expand to receive the contents of the ventricle, but act more like rigid tubes; the result of which is, that during the cardiac systole the inflow into the vessels, which is always greater than the outflow at that period, produces a continuous rise in arterial pressure during the whole time of systole. The point of highest pressure of pulse wave, or, in other words, the highest part of the pulse tracing, is thus thrown toward the end of the ventricular part of the pulse curve, or, in other words, closer to the dicrotic notch, *d*, which marks the end of the systole. In other words, in cases where the larger arteries are not fitted to contain the quantity of blood contained in the ventricle, the latter forces the blood at first against a comparatively weak resistance, which, however, goes on increasing very rapidly as the large arteries become gradually more and more tensely filled; and the pressure within these latter necessarily rises from the commencement to the end of the cardiac systole. This, then, is the reason why in such circumstances the highest part of the pulse curve is nearest our line *d*, or the dicrotic notch, which corresponds to the end of the ventricular systole.

I have as yet said nothing of the indenture (*c*) which precedes the dicrotic notch, and which, on that account, is usually

described as the pre-dicrotic notch. The exact significance of this notch is still by no means so fully understood as is desirable. It would seem that its appearance results from the fact that, at the moment when the aortic valves are forced open, the column of blood contained in the aortic arch and larger branches receives a sudden impulse towards the periphery, and the inertia of this column of blood, thus set in comparatively rapid motion, produces a negative wave at the commencement of the aorta, which is propagated towards the periphery in the same manner as the positive wave which preceded it. I have spoken of the causes which may theoretically produce anacrotism, and also the probable cause of the pre-dicrotic notch, and must now proceed to refer to the conditions under which, in practice at the bedside, we find the ventricular part of the pulse wave so modified.

If the glottis be closed, and the pressure within the thorax and abdomen be raised by powerful continuous contraction of the respiratory muscles, we produce a change in the distribution of the blood in the arteries and veins. The intra-abdominal and intrathoracic veins are relatively empty, and an abnormally large quantity of blood accumulates in the systemic arteries. During this state the arterial walls are more or less powerfully distended, and, following known laws regarding arterial elasticity, they are in that condition more rigid than when their calibre is normal. Even in tolerably young subjects, by this means we can easily produce artificially an anacrotic pulse wave, the arteries being rendered relatively rigid in relation to the quantity of blood which is forced into them at each ventricular contraction. This arterial engorgement or high pressure, only temporary in such an experiment, is, however, lasting in certain diseased conditions, the most marked of these being the arterial high pressure which accompanies certain forms of chronic kidney disease, in which latter case the conditions are still more favourable for the production of an anacrotic pulse wave, seeing that not only are the arteries abnormally rigid from the distension, but also that the quantity of blood forced into them

with each contraction of the ventricle is relatively and absolutely great, owing to the existence of excentric hypertrophy of the left ventricle. Analogous conditions occur, as already indicated, in cases of atheroma or calcification of the larger arteries, such as occur in old age. The conditions, therefore, which produce the anacrotic pulse wave are in practice either abnormal distension of the larger arteries, accompanied or not by hypertrophy of the ventricle, or rigidity of the arterial walls due to changes in molecular structure of their middle coats. The more marked these conditions are, the more is the second elevation (*c*) higher than the first (*b* in figs. 16 and 17). In practice, all imaginable intermediate forms between the typical pulse wave of health and the typical anacrotic pulse wave, as in fig. 17, are encountered, and it is usually easy in each individual case to tell from the other phenomena whether the anacrotism be due to simple distension of the arteries from high pressure, to molecular change in the arterial coats, or to hypertrophy of the heart. From what I have said, it will be understood that, although the anacrotic pulse wave very often means an abnormally high arterial pressure, this is by no means always the case. Finally, before leaving the changes confined chiefly or entirely to what I have named the ventricular part of the pulse wave, a word may be said regarding the conditions which favour the appearance of a well-marked pre-diastolic notch. The condition fitted to produce this notch in its most marked form, is that in which the part of the systemic arteries nearest the heart is abnormally rigid; for it need scarcely be said that if this latter part of the systemic arterial system is fairly elastic, it will contract behind the suddenly impelled first wave, and prevent more or less completely the formation of a negative wave or tendency towards a vacuum at the commencement of the aorta.

We now turn to consider a different series of changes in the form of the pulse wave, which are due to changes in the arterial circulation of an entirely different kind from those above referred to, and in which practically invariably the dicrotic notch is abnormally exaggerated.

*Abnormally Dicrotic Pulse Waves.*—I have said above, that on closing the glottis and contracting powerfully the respiratory muscles, the systemic arteries are at first abnormally filled with blood; this abnormal distension very soon, however, gives place to an abnormal emptiness of these vessels, owing to the fact that the pressure on the intrathoracic veins diminishes the quantity of blood which reaches the ventricle, the result being that the blood accumulates chiefly in the veins of the head and limbs. The artificial arterial anæmia so produced leads to a characteristic change in the form of the pulse wave, which becomes, as in fig. 18, smaller in size and more markedly dicrotic than even the normal pulse, while all trace of anacrotism completely disappears. It is unnecessary for us to go minutely into the theory of the production of the abnormally dicrotic pulse wave. For practical purposes it will suffice to refer to the conditions which lead to the appearance of this form of curve. Roughly speaking, these may be said to consist in abnormal emptiness of the arterial system, such as is produced, for example (a) by anæmia after venesection, in which case the absolute quantity of blood in the arteries is diminished, although these latter contain relatively normal amount; (b) in cases of unusual expansion of the arterioles and capillaries leading to a relatively rapid outflow from the arteries, as in the condition produced by amyl-nitrite inhalation; or finally, dicrotism may be produced by (c) diminution in the quantity of blood which enters the aorta through the ventricle—the most marked examples of which are to be found in cases of uncompensated mitral regurgitation.\* Such are the conditions which, in practice, are found to produce the dicrotic pulse wave; and it may be noted in passing, that simple



FIG. 18.  
Abnormally Dicrotic  
Pulse Wave.

\* Not unfrequently two or more of these causes may be combined, as happens in fevers.

or pure dicrotic pulse wave invariably results from abnormally low arterial pressure, the cause of which, in individual cases, it is rarely difficult to discover.

*Hyperdicrotic* is the term applied to that form of the dicrotic pulse in which the dicrotic notch descends lower than the commencement of the systolic rise. This is due to the fact that each successive cardiac systole follows its predecessor before the pressure within the artery has fallen below that which it presented at the dicrotic notch. This form of curve (fig. 19), although presenting a notch in its ascending part is due to entirely different conditions from those which produce the true anacrotic pulse wave with which it can never in practice be confounded, owing to the fact that the rounded smooth eminences of which it is made up show it at a glance to be of the dicrotic type; it is, in fact, an exaggerated dicrotic pulse wave.



FIG. 19.  
Hyperdicrotic Pulse Waves.

The anacrotic and the dicrotic pulse waves are the two principal simple modifications which are met with, but there are various intermediate or combined pulse waves due to combination of the conditions fitted to produce the anacrotic and the dicrotic waves,—for example, in cases of aortic regurgitation with hypertrophied ventricle, the first or ventricular part of the curve usually is of the true anacrotic type, while on cessation of the systole, the reflux into the heart causes a more or less powerful negative wave producing an abnormally deep dicrotic notch. In addition to this the rapid filling of the comparatively empty arteries with each ventriculatory systole leads to an abnormally steep and high ascending limb of the curve.

We have given the conditions which lead to the two principal forms of pulse wave met with in disease; but as I have said, all possible combinations of these conditions are constantly occur-

ring, leading to some less well-defined type of pulse curve. Into a detailed account of these more complicated pulse curves we cannot enter here. What we have already said will enable the observer to understand the meaning of each. Examples of these are found in the various modifications of pulse curve in prolonged fever cases, in the early stages of which it is often high, bounding with a tendency towards the anacrotic type, but gradually, from day to day becoming more and more dicrotic, and not unfrequently being eventually hyperdicrotic.

**Cardiograph.**—The results which may be obtained by the use of the cardiograph are on the whole less satisfactory than those of the sphygmograph. This is in part due to the fact that none of the instruments at present in use for recording the contractions of the human heart can compare with the sphygmograph in so far as compactness and accuracy are concerned. While some observers, such as Landois, have attempted to record the form of the apex-beat by applying to the surface of the chest the sphygmograph of Marey, the majority of the instruments employed at the present day are transmission instruments, being constructed in the same manner as the transmission sphygmograph, which I have already described. It is extremely desirable that we should have at our disposal some direct acting cardiograph similar in principle to the sphygmograph of Chauveau and Marey. Such, however, has not as yet been described. In the meantime, the transmission cardiograph is the one which is almost universally employed. That of Burdon Sanderson,\* or the polygraph of Marey,† are amongst the best, if not the very best. I need not enter upon the manner of using these and similar instruments, but will proceed to describe, firstly, the typical normal heart curve; and secondly, the principal modifications which it may present.

\* "Handbook of the Physiological Laboratory," edited by Burdon Sanderson.

† *Loc. cit.*

*Normal Heart Curve.*—In fig. 20 is represented a typical normal curve of this kind. The curve, it will be seen, immediately after rising from its lowest point *f*, describes a more or less well marked rounded elevation between *f* and *a*, and from *a* it ascends at first rapidly, afterwards somewhat more slowly, to its highest point *b*, from whence it describes a more or less obliquely descending, usually undulating line to *e*, after which the curve descends, at first slowly, then more rapidly, and

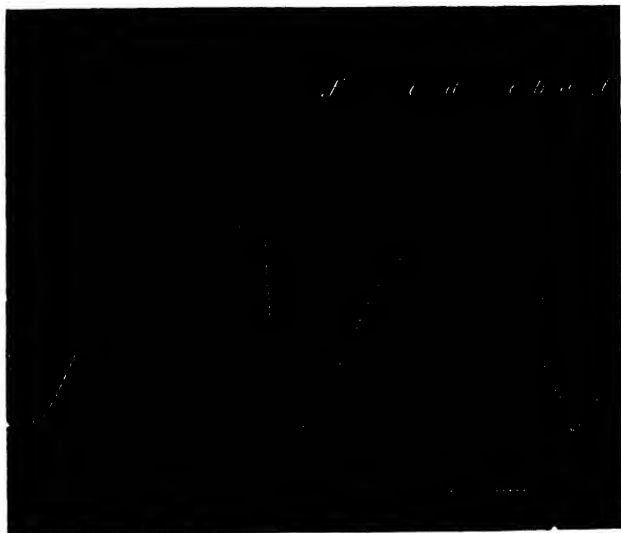


FIG. 20.—Normal Heart Curve.

finally with increasing slowness, until the point *f* is reached. That part of the curve lying between *f* and *a* corresponds in time to the contraction of the auricles, and when the curve is taken from the apex, the elevation between *f* and *a* is due to the more or less sudden filling of the ventricles, which results from the auricular contractions. That part of the curve lying between the lines *a* and *e* is produced during the time of contraction of the ventricular muscle, while the part from *e* to *f* corresponds with the passive expansion of the ventricular



muscle. In so far as the ventricles are concerned, we may divide the whole heart curve into two parts—viz., first, that from *a* to *e*, during which the ventricular muscle is in a state of contraction; and second, that from *e* to *a*, which corresponds to the ventricular diastole. The sudden rise from *a* to *b* is produced by the tightening of the ventricles over their contents, and the point *b* corresponds in time to two important phases of each heart beat—viz., first, the moment of closure of the auriculo-ventricular valves; and secondly, the moment when the heart muscle has fairly grasped its contents. The height of *b* over *e* gives some indication of the difference in antero-posterior diameter of the heart at commencing systole as compared with the end of the systole, for it need scarcely be said that the larger the quantity of blood contained in the ventricle at the commencement of the ventricular systole, the greater will be its antero-posterior diameter, and therefore the more powerful impulse will be given to the chest wall and cardiograph button. As the heart empties itself during systole the antero-posterior diameter of the ventricles diminishes with corresponding rapidity, and the pressure against the chest wall and cardiograph button falls in the same ratio. The result of this is that, *cæteris paribus*, the degree to which the line joining *b* and *e* descends gives a valuable indication regarding the quantity of blood thrown out by the ventricles at each systole. The meaning of the notches *c* and *d* is not satisfactorily understood. This much, however, is certain—viz., that they are not due to inertia vibrations of the recording lever, as has been asserted by some; and also, that in not a few cases the form of the curve lying between the lines *a* and *d* of the cardiogram resembles very closely that lying between the line *a* and *d* of our normal pulse curve (fig. 16). In other words, they are probably due to oscillations of the column of blood contained in the ventricles and larger arteries. The notch *d*, when well marked, corresponds to the conclusion of the outflow of blood from the heart, and is therefore the analogue of the dicrotic notch of the pulse wave. It must be added, however, that it

is by no means uniformly to be seen. Of greater importance is the position of the last elevation or corner of the curve at *e*, which can in almost all curves clearly be made out. This elevation marks the commencing relaxation of the ventricular muscle, and by measuring the distance between the lines *a* and *e* in the manner which will be described in a note appended to this chapter, we are enabled to learn with absolute accuracy the duration of the ventricular systole in any given case. I must mention, in passing, that the duration of the ventricular systole, and the duration of the outflow from these cavities, by no means necessarily or even usually correspond. The ventricular muscle contracts with a certain definite force, and remains contracted for a certain definite time, neither of these being influenced by the quantity of blood contained in the ventricle at the commencement of its contraction. The result of this is, that where a very small quantity of blood is contained in the ventricles at the commencement of their contraction, the outflow from them may have concluded some tenths of a second before the ventricles begin to relax. The distance between the lines *e* and *f* gives some indication of the rapidity with which the heart muscle has relaxed after the conclusion of its contraction. Where the elasticity of the heart muscle is modified, as when the blood contains a largely diminished quantity of oxygen, the ventricular muscle takes a longer time to relax than is normally the case, and the curve from such a beat descends less rapidly than in health.

The cardiographic curve then enables us to measure with very considerable accuracy the absolute and relative duration of the different phases of the cardiac revolution. It also gives us some idea of the force of the ventricular contraction corresponding to the height of the line *a* to *b*, and it further affords valuable information regarding changes in the force and frequency of the heart's action, which make up the different forms of irregularity of the heart.

It is unnecessary to refer more in detail to the normal typical heart curve, and I turn now to mention those diseased

conditions which modify its form; and first, with regard to the cardiogram in aortic regurgitation. After what has been said regarding the meaning of the various parts of the normal heart curve, it is not difficult to understand in what way these may be modified in a typical case of aortic regurgitation. In the first place, the ventricle, before the contraction of the auricles, is abnormally distended with blood; and on the auricles propelling their contents into the already filled ventricle, an abnormally great distention of the ventricles occurs. The result of this is that, in cases where there is no failure in the power of the auricular walls, the elevation between the letters *f* and *a* is abnormally high. On ventricular contraction occurring, the antero-posterior diameter of the heart diminishes very rapidly, corresponding with the abnormally large quantity of blood contained in the ventricle, so that the line joining points corresponding to *b* and *e* is unusually steep, while the regurgitation of blood through the incompetent aortic valves, after the cessation of the systole, causes a dilatation of the relaxing cardiac muscle sufficient to produce, in most cases a very well marked rise after *e*. It is important to note that, in the heart curve of well marked aortic regurgitation, it is often impossible to find the exact point corresponding to *e* in the normal curve at which the systole suddenly ceases. The corner of the curve preceding the descent is usually in the aortic regurgitation cardiogram some fraction of a second later than the time of commencing relaxation. In all, or nearly all, cases of aortic regurgitation, the heart curve presents two well marked peaks, and this may be said to be the distinguishing character of the cardiogram of that disease, and, roughly speaking the more marked this bicornual character is, the greater is the incompetence of the valve. Such is the curve when the ventricular muscle is comparatively unimpaired in contracting power, as, for example, in sudden rupture of one of the cusps of the valve, or when one of these is artificially destroyed in the lower animals; but where the ventricle no longer completely empties its contents at every contraction, the fall of

the line from *b* to somewhere about *e* becomes, as we might anticipate, less and less steep, due, it need scarcely be said, to the slighter diminution in the antero-posterior diameter of the ventricles, which occurs when the ventricle no longer empties itself completely at each contraction. In these cases, then, the bicornual character is not so well marked as is otherwise the case in that disease, but still it is usually sufficiently recognisable.

With regard to the curve in cases of mitral incompetence, we would anticipate, where this condition was well marked, that the heart curve would be modified chiefly, if not exclusively, at that part which corresponds in time to the closure of the auriculo-ventricular valves; in other words, at the point marked *b* in fig. 20; and this to a certain extent is the case. As a rule, we find the ascending line from *a* to *b* less steep than is normally the case, and the peak at *b* rounded off to some extent. This is apparently the only characteristic change in the form of the heart curve which results from simple mitral incompetence; but it is by no means usually well marked, owing to the fact that the large quantity of blood which leaves the ventricle during systole causes a very considerable diminution in the antero-posterior diameter during ventricular systole; so that the line from *b* to *e* is unusually steep, thus tending to cover the rounding off which the peak *e* would otherwise present. It is rare to find that the reflux of blood from the auricle into the ventricle, which follows the conclusion of the systole of the ventricle, distends the latter with sufficient force to cause, as is the case in aortic regurgitation, second elevation after *e*. Finally, where the auricles are hypertrophied, the auricular elevation in the heart curve of mitral incompetence is abnormally well marked. Such are the changes produced by simple mitral incompetence, which has been more or less completely compensated by hypertrophy of the auricle and dilatation and hypertrophy of the ventricle; but in cases where either the auricle or the ventricle begins to fail, there are endless modifications, which what has above been said regarding the meaning of the different parts of the normal heart curve

will enable the physician readily to understand ; but it should be added, that in advanced mitral cases, where irregularity is a pronounced element of the case, the heart curve becomes so *bizarre* in form that it is difficult, and sometimes impossible, to understand what is the meaning of the different elevations which it presents.

With regard to pure mitral stenosis, we would *à priori* expect that the ascending line from *a* to *b* would be abnormally steep, owing to the abnormal rigidity of the mitral valve, and that the peak *b* would be unusually sharp, corresponding as this does with the thump which is characteristic of the disease in question — and such is certainly sometimes the case ; but when it is remembered how exceedingly rare is stenosis of the mitral valve uncomplicated with regurgitation, it need cause no wonder to find that the cardiogram in mitral stenosis is by no means characteristic or typical. Still, the curve in all, or nearly all, cases presents sufficiently well marked deviations from the normal which are fitted to throw much light upon the condition of the heart in individual instances ; and careful attention to the period of time in the heart revolution at which these abnormalities take place readily indicates their meaning.

There are many modifications of the heart curve which it is impossible to describe here in detail ; and indeed, in practice, almost every imaginable combination of the abnormalities above described is encountered.

**Sphygmomanometer.** — An instrument has recently been devised and introduced by von Basch,\* to which he gives the above name, for the purpose of estimating the blood-pressure in the human subject, and which has undoubtedly considerable clinical value. In principle it closely resembles an apparatus previously described by Prof. C. S. Roy and myself,† which

\* *Zeitschrift f. kl. Med.*, vol. ii., 1881, p. 79 ; and vol. iii. p. 502.

† *Verhandlung der physiol. Gesellschaft zu Berlin*, 15th Feb. 1878. *Journal of Physiology*, vol. ii. p. 323. \*

was more specially adapted for estimating the blood-pressure in the lower animals. In Von Basch's sphygmomanometer, a small cushion of membrane is made to press upon the skin over the radial artery, and the pressure is communicated through water to a column of mercury, by which its value can be ascertained. The pressure of the cushion upon the radial artery is gradually increased, until all pulsation in the vessel beyond the constricted point ceases; and this point is taken as the maximum arterial pressure.

While open to many sources of error, the readings of this instrument, if taken with sufficient care, appear to give results which are approximately accurate; at any rate, quite sufficiently so for ordinary clinical work. In healthy men between the ages of twenty and sixty the pressure averages about 150 m.m., but may be as low as 135 m.m., or as high as 170 m.m. High pressures much exceeding these, and running as high as 245 m.m., are met with in such affections as chronic Bright's disease with cardiac hypertrophy, while in such diseases as anæmia, phthisis, &c., the blood-pressure is found to be very low.

This instrument seems to be specially suited for watching the progress of individual cases from day to day, and in particular for observing the effects of treatment upon the blood pressure.

#### *Note on the Measurement of Tracings.*

In order to measure the relative duration of different parts of the pulse wave (and the remarks which follow apply equally to heart curves), a certain method is necessary to correct the error, due to the fact that the point of the lever describes, not a straight vertical line, but the segment of a circle. The most convenient method of doing this is as follows:—Having obtained a satisfactory curve from the radial artery, the sphygmograph is

removed from the arm, and the paper in its holder is again passed through the clockwork, the point of the lever being fixed so as to draw a straight abscissa line—i.e., one parallel to the line of the movement of the paper. This line may be drawn either below the curve or through it; but where absolute accuracy is desired, it ought to be at the level of the centre of that circle of which the lever describes a segment when the paper is at rest. The paper is then either drawn backwards or put a third time through the clockwork, and successive parallel curved lines are described by the lever point connecting those portions of the curve which it is desired to measure with the abscissa line. In fig. 16 there are certain dotted lines, of which the horizontal represents the abscissa line, and the vertical curved lines represent segments of the circle described by the recording lever. Since the movement of the clockwork of the sphygmograph is during the time when any single pulse curve is being recorded, fairly uniform, and since the rapidity of the heart beat is readily learned by the watch, it is not difficult to say what fraction of a second corresponds to the distance between any two of our vertical curved lines. For example, in fig. 16 the pulse was, let us say, sixty in the minute; and as the distance from the vertical lines *a* to *c* is roughly one-third of the whole, we know that the duration of that portion of the pulse wave is equal to one-third of a second. In the same way, the duration of any other portion of the pulse wave may be determined with sufficient accuracy. This method enables us to learn the *duration* of any of the phases of the pulse curve, while the relative importance of the various parts of the same curve—that is, the tension of the blood in the artery at different points—is represented by the height of the tracing as measured from a horizontal line running through the lowest part of the pulse wave.

## CHAPTER XIV.

### Respiratory System.

THE first symptoms which require notice in connection with the respiratory system are—

**Subjective phenomena**, such as pain, tickling, burning, &c., which are frequently felt over the larynx, trachea, and bronchi, when the structures are the seat of disease, and are usually aggravated by pressure, and by the acts of speaking and coughing. *Pain* may manifest itself in connection with disease of the lung tissue, but it attains its greatest importance in cases of pleurisy, where the pain has a peculiar dragging, shooting character, is increased by pressure, and by any movement of the thorax. Its differential diagnosis is very important.

We must distinguish the pain of pleurisy—

(1.) *From the Pain of Pleurodynia*, or rheumatism of the intercostal muscles. In this condition the pain usually comes on with excessive suddenness, after some abrupt movement, and is unaccompanied by pyrexia, or by friction sound.\*

(2.) *From the Pain of Intercostal Neuralgia*.—In this affection there are commonly three tender points (*points douloureux* of Valleix) in the course of the affected nerve, one close to the vertebral column, one in the axilla, and a third over the terminal branches near the sternal border. The presence of these

\* It must be remembered, however, that this auscultatory phenomenon may be wanting in the early stage of pleurisy, so that the physician may have to refrain from a positive diagnosis until this symptom has had time to develop.



points the neuralgic character of the pain, and the absence of all pulmonary physical signs, except such alterations of the respiration as the pain occasions, will suffice to distinguish this affection from pleurisy.

(3.) *From the Pain of Herpes Zoster.*—This eruption, which follows the course of the intercostal nerves, is sometimes preceded by severe pain, usually of a burning character. The marked cutaneous hyperæsthesia which frequently accompanies this pain will suffice to distinguish it from pleurisy.

(4.) *From the Pain of Periostitis and other Surgical Affections of the Thoracic Wall.*—Careful examination of the ribs will make clear the nature of such pain.

**Breathing** will be more conveniently considered hereafter (p. 183).

**Cough.**—The removal of foreign substances from the respiratory passages is effected by means of the acts of sneezing and coughing,—two forms of explosive expiration which are both, as a rule, excited reflexly, and which both consist in a closure of the respiratory passages after a deep inspiration, followed by a sudden, forcible, and noisy opening of the same, the result of a powerful expiratory effort. In the case of sneezing, the closure is effected by the pressure of the soft palate, by means of the tongue, on the posterior wall of the pharynx, while in coughing the closure takes place at the glottis.

Coughing is excited by irritation of the terminal branches of the superior laryngeal nerve distributed to the mucous membrane of the larynx and trachea. The inhalation of cold air, or of air laden with dust, the passage into the larynx of particles of food, or other foreign bodies, and the collection of secretions, or of such morbid productions as blood, or pus, all tend to excite coughing, which is more liable to occur when in addition there is hyperæsthesia of the parts, the result of catarrh or inflammation. The terminal branches of the vagus distributed on the bronchi, lung tissue, pleura, or abdominal viscera, or even the small branch to the auditory

meatus, may be the starting point of the irritation, while in sensitive individuals, the action on the skin of a draught of cold air is sufficient to set up cough. Anæsthetic conditions of the larynx are occasionally met with in which such local irritations as those mentioned are not sufficient to excite cough; and depression of the activity of the reflex centre in the medulla, the result, for example, of the accumulation of carbonic acid in the blood, or of the action of opium, may diminish or completely abolish the act of coughing, and thereby cause a dangerous accumulation of secretion in the air-cells.

In examining cough as a symptom, it is well to note—

*1st. Its Frequency and Rhythm.*—The physician should inquire whether it comes frequently, each individual cough being separated by a tolerably constant interval, or whether there occur paroxysms of coughing with intervals of quietness.

*2nd. Its Character.*—This may vary very greatly. The cough may be dry, as in pleurisy, the first stage of phthisis, &c., or moist, as in chronic bronchitis, and in the last stages of phthisis. It may be painful, as in acute pleurisy, and the patient then instinctively tries to suppress the cough which gives him so much pain; and this short, dry, suppressed cough is frequent at the commencement of acute pneumonia, and also in cases of intercostal neuralgia, pleurodynia, pericarditis, and peritonitis. Very different from this is the loud barking cough of hysteria, which is obviously produced at will, and calculated to attract the utmost amount of attention. In laryngitis, even when the disease is very slight, the cough is hoarse, husky, stridulous, and croupy in character; and when much submucous infiltration has taken place, or if there be extensive formation of false membrane, the act of coughing is almost noiseless. The hard metallic laryngeal cough met with in cases of aortic aneurism, where the recurrent laryngeal nerve is interfered with, is often of considerable diagnostic value.

*3rd.* Notice whether the cough is obviously brought on by such causes as exertion, change of posture, inhalation of cold air, of dust, or of irritating chemical vapours.

**4th.** Notice if the paroxysm terminates in a fit of vomiting, as so often occurs in whooping cough, phthisis, and chronic bronchitis, or in the prolonged, clear, shrill inspiration which characterises the first of these affections.

**Sputa.** — In almost every affection of the respiratory organs, more or less expectoration follows the act of coughing. Occasionally, however, this is absent; and in the case of young children, even when the cough is accompanied with expectoration, the sputum is swallowed as soon as it reaches the mouth. It must be borne in mind that the material coughed up may not come originally from the respiratory tract: for secretions from the mouth, nose, and pharynx may pass the rima glottidis, and, irritating the mucous membrane of the larynx, be coughed up again. Bleeding from the posterior nares may thus simulate hæmoptysis.

*Chemical Characters.*—As yet the chemical analysis of sputa has not proved of much diagnostic value. Consisting, in the main, of water, sputa have at different times been found to contain serum, albumen, paraglobulin, paralbumen, myosin, nuclein, glycogen, various fatty acids, leucin, tyrosin, &c., in addition to the mucin which is invariably met with even in the healthy state, and which imparts to the expectoration its peculiar viscid character. Albumen is always present when there is inflammation of the air-passages or lung-substance. In cases of diabetes, sugar has been detected in the sputa, and in renal affections urea may sometimes be found.

#### *Macroscopic Characters of Sputa.\**

For purposes of ready description the various varieties of sputa may be classified as follows, each being named after its principal constituent.

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\* In all cases of laryngeal and pulmonary disease the sputa should be regularly examined, and for this purpose the expectoration for twenty-four hours should be collected in a glass vessel of such shape as to permit of rapid and satisfactory inspection.

(1.) *Mucous sputum* is transparent, clear, and glassy, and has a viscid and ropy consistence which is best appreciated by pouring it from one vessel into another. It is sometimes present in health, often becoming constant in advanced life, but is most frequently found in the earlier stages of bronchial catarrh. There is a very slight admixture of pus corpuscles.

(2.) *Muco-purulent sputum* may occur in almost every affection of the bronchi and lung. When allowed to stand in a vessel, the pus corpuscles sink to the bottom, leaving the clear mucus floating on the surface. Sometimes, however, a more intimate mixture of these two elements takes place. When there are present cavities in the lung, the sputum often takes peculiar forms. Round flattish masses of purulent matter, with well defined margins, are then seen lying at the bottom of the vessel. From the resemblance of these masses to coins this variety of sputum is sometimes called nummular. Very rarely a similar appearance is observed in cases of chronic bronchitis.

(3.) *Purulent sputum* resembles closely ordinary pus as obtained from an abscess. It has the same yellow opaque appearance, and separates into two layers when allowed to stand, the lower being composed of pus corpuscles, the upper of plasma. This variety of sputum is usually derived from suppurating cavities in the lung, or is the result of other collections of pus (for example, empyæma) bursting into a bronchus.

(4.) *Serous sputum* is that form which is met with when copious transudation takes place from the pulmonary circulation, as in œdema of the lungs. It has a characteristic thin, transparent appearance, and is usually copious and frothy. Consisting as it does, in great measure, of transuded serum, this form of sputum contains much albumen. This becomes at once apparent when the filtered sputum is slightly acidulated with acetic acid and then boiled.

(5.) *Sanguineous sputum*.—The sputum may be simply streaked with blood (as in the early stages of phthisis, &c.), or the blood may be mixed intimately through the mass. This latter form is that most usually met with in the later stages of

phthisis, in cases of hæmorrhagic infarction, and in lobar pneumonia. In the last-named affection the sputum is of a rusty colour, due to chemical alteration of the blood pigment, and this may pass into citron-yellow and green. It has been already said that blood from the throat and posterior nares may trickle into the trachea and be coughed up. The primary source of the hæmorrhage is then, however, usually clear. It is more difficult to distinguish hæmorrhage from the lungs (hæmoptysis) from that from the stomach (hæmatemesis). The history and physical examination, and the nature of the act by which the blood reached the mouth, will help greatly towards diagnosis; but it must be remembered that the blood coughed up may be swallowed and then vomited. In hæmoptysis the blood is usually bright red, fluid, and frothy, has an alkaline reaction, and when examined microscopically is found to contain more or less of those cellular elements which are peculiar to the respiratory tract. In hæmatemesis the blood is dark and venous, sometimes chocolate-brown, resembling coffee-grounds, often clotted, free from froth, acid in reaction, and when microscopically examined is found to contain fragments of food. This subject has been already considered in reference to hæmatemesis.

#### *Physical Characters of the Sputa.*

(1.) *Quantity.*—The amount of expectoration may vary very much, and this indication may become of considerable diagnostic value, as, for example, when in the course of some acute affection (bronchitis, pneumonia) the scanty sputum suddenly becomes more abundant and more readily expectorated, showing thereby that the acuteness of the inflammation is subsiding. In bronchiectasis very large quantities of sputum are brought up at one time, and so marked is this symptom that it may suffice in many cases to establish a diagnosis in the absence of other signs.

(2.) *Form and Consistence.*—The more mucus the sputum contains, the firmer will be its consistence, and the more distinct its form. Tenacious sputa are consequently found in the acute

stage of bronchitis, pneumonia, phthisis, &c. In the absence of mucus, the sputa lose their individual shapes, and, when collected in a vessel, they coalesce with each other. Such is the case with the purely purulent and the serous sputa. Tough sputa from phthisical cavities preserve their flattened, coin-like (nummular) shape after expectoration—an indication of some diagnostic value.

(3.) *Smell*.—As a rule, sputa are devoid of any very marked odour. When, however, putrefaction becomes developed, the odour of the breath and of the expectoration becomes most overpowering. This occurs to a marked degree in bronchiectasis, putrid bronchitis, and pulmonary gangrene.

(4.) *Colour*.—To the yellow or yellow-green tinge which is imparted to the sputum by pus cells when they are present, allusion has already been made. The red colour of sanguineous sputa has also been described, passing into rust-colour, yellow, saffron, and finally green, as the hæmoglobin becomes more and more highly oxidised. Löwer has pointed out that in very hot weather a similar yellow tint is sometimes to be seen in the sputum, resulting from the development of a fungus (*leptothrix*). A yellow or a green discoloration frequently appears in the sputa in cases of jaundice, due to the presence of bile pigment; and those who are much exposed to smoke, or who work in coal mines, frequently expectorate the carbonaceous particles which they have inhaled, to such an extent as to blacken the whole of the sputum.

#### *Microscopic Examination of the Sputa.*

(1.) *Pus, Blood, and Mucus Corpuscles*.—The recognition of these corpuscles is very readily made by means of the microscope. What diagnostic significance attaches to each has been already stated, and does not demand further remark.

(2.) *Epithelial Cells*.\*—The ordinary pavement epithelial

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\* These structures are best seen when stained with methyl-anilin. A small particle of the sputum should be mixed on the slide, with a drop of a watery solution (1-1000) of that pigment.

cells of the mouth are almost invariably present in sputa, becoming mixed with the expectoration on its passage through the mouth. They are of large size, polygonal in shape, finely granular, and possess a large, refractive, ovoid nucleus.

The columnar epithelium of the bronchial mucous membrane almost never appears in the sputa. When ciliated cells *are* present, they usually come from the nasal cavity.

Much more important for diagnosis is the occurrence of the epithelium of the pulmonary alveoli. As Friedländer\* has shown, these alveolar epithelium cells never appear in the sputum in their normal flattened condition, but invariably swell up and become spherical when brought into contact with liquid, or when they undergo inflammatory change. In the sputa, this alveolar epithelium is readily recognised. The cells are spherical or slightly oval, have a diameter two to four times greater than that of a leucocyte (thus distinguished from all other round cells in the sputum), contain granular protoplasmic masses, and possess one or more large oval nuclei with distinct nucleoli. They further differ from all other cells to be found in the sputum in that they readily become pigmented, and undergo fatty and myelin† degeneration,—changes which the other varieties seldom or never show. Bearing these points in view, the distinguishing of the alveolar epithelium can seldom be a matter of difficulty.

Regarding the diagnostic value of the cells,‡ it is important to observe that above the age of thirty to thirty-five years alveolar epithelium is to be found in the sputa of perfectly healthy persons, but that variety of cell is not found in individuals whose age is below thirty. At all ages, however, alveolar epithelium may be found in the sputa of many affections of the

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\* "Ueber Lungenentzündung nebst Bemerkungen über das normale Lungen-epithel." Berlin, 1873.

† Panizza (*Deut. Arch.*, vol. xxviii., 1881, p. 343) argues strongly in favour of the view that this myelin transformation is mucous degeneration.

‡ On this subject see the very careful paper by Guttman and Smidt in the *Zeitschr. f. klin. Med.*, iii., 1881.

respiratory organs—in œdema, hypostatic congestion, hæmorrhagic infarction, pneumonia, and in all the forms of phthisis. In simple bronchial catarrh of individuals under thirty, no alveolar epithelium is to be found in the sputa, unless the very finest bronchioles be affected, and then these cells appear only in small number. In commencing phthisical catarrh of the apex, however, alveolar epithelium is to be found in considerable quantity long before any physical sign can be detected, and in young individuals in whom all the other causes mentioned may be excluded, the occurrence of alveolar epithelium is almost certainly diagnostic of commencing phthisis.

(3.) *Debris of Lung Tissue*.—In any disease which involves destruction of lung tissue, we may find in the sputum the elastic fibres which had formed the framework of the broken-down alveolar walls. These fibres may be distinguished under the microscope without difficulty. They usually lie in groups coiled and twisted, sometimes recalling by their arrangement the outline of the alveoli. Their dichotomous branching and well-defined double contour, and still more, their resistance to the action of caustic alkalis, make their recognition a matter of little difficulty. It is well, as Fenwick recommends, to boil the sputa with caustic soda until the mixture becomes thin and watery. The elastic fibres will then readily sink to the bottom of a conical glass, and can be secured by means of a pipette.

While the debris of lung tissue occurs by far most frequently in the sputum of phthisis, it may also be found in cases of pulmonary abscess and of gangrene of the lung. In the last-named affection the lung tissue is only to be found in very fresh sputum. It rapidly disappears, being apparently acted upon and dissolved by some peculiar ferment which is present in the expectoration in such cases. It need hardly be said that where such elastic fibres occur, we have absolute proof of the destruction of lung tissue, hence the great importance of this symptom in case of phthisis where the physical signs are not distinct.

(4.) *Fibrinous Bronchial Casts*.—In pneumonia and in croupous



bronchitis, there are to be found in the expectoration casts in fibrin of the finer bronchioles and their branches. In the sputum they are usually rolled together, and only unroll and spread out when washed with water. The perfect way in which they reproduce the arrangement of the bronchioles makes the recognition of these casts easy. In pneumonia they are most numerous in the sputum on the third and fourth day of the affection, and Remak, who devoted much attention to the subject, has pointed out that the earlier these casts appeared, and the greater their quantity, the more quickly will recovery set in, and the more completely will the affected lung recover from the disease.

(5.) *Crystals* are occasionally met with in sputa, the most common being the long, fine, colourless, needle-shaped crystals of the fatty acids. They have some superficial resemblance to elastic fibres, but are easily distinguished by the fact that they dissolve at once in ether, a reagent which does not affect elastic fibre. These fatty acids are found in cases of putrid bronchitis, bronchiectasis, and pulmonary gangrene.

Another variety of crystal which may be found in the sputum are those usually known as *Charcot's crystals*, after the name of their discoverer. Their exact nature is a matter of some doubt. In shape they vary somewhat, but are usually long, fine, sharp, and spindle-shaped; they are colourless, are insoluble in alcohol, but are readily dissolved by acid or alkalis. Charcot's crystals occur most frequently in asthma, and have been supposed to be the exciting cause of the paroxysm, by irritating the terminations of the vagus.

Other crystals, such as cholesterin, hæmatoidin, leucin, tyrosin, oxalic acid, and triple phosphate, occur in the sputum, but do not demand special notice here.

(6.) *Micro-organisms* of various kinds may be found in the sputum, such as *leptothrix*, *oidium albicans*, and, rarely, *sarcina*. Bacteria and vibriones are very frequently to be seen in the sputum of gangrene and bronchiectasis.

By far the most important of these micro-organisms, how-

ever, is the *tubercule-bacillus*, which Koch has recently\* discovered. These bacilli are delicate, rod-shaped structures, in length usually about one-fourth or one-third the diameter of a blood corpuscle, and are motionless. They can only be detected after careful staining; and for this purpose several methods may be employed, two of which will be detailed here.

(a.) *Ehrlich's Method*.—A thin layer of sputum is spread on a cover-glass, which is then gently heated over a flame for a few seconds to coagulate the albumen, and placed in a staining solution prepared as follows:—Five cubic centimetres of pure anilin are added to 100 cubic centimetres of distilled water, well shaken and filtered, and to the filtrate a saturated alcoholic solution of fuchsin or methyl-violet is added until precipitation commences. The cover-glass is allowed to float on this for half-an-hour. It is then washed in a solution of nitric acid (1 to 2), and afterwards in distilled water. In this way the stain is extracted from everything but the bacilli.

(b.) *Heneage Gibbes' Method*.†—This process is to be preferred for clinical purposes, as it is rapid and does not require the use of nitric acid. This staining solution is made as follows:—“Take of rosanilin hydrochloride two grammes, methyl-blue one gramme; rub them up in a glass mortar. Then dissolve anilin oil, 3 c.c., in rectified spirit, 15 c.c.; add the spirit slowly to the stains until all is dissolved, then slowly add distilled water, 15 c.c.; keep in a stoppered bottle. The sputum having been dried on the cover-glass in the usual manner, a few drops of the stain are poured into a test-tube and warmed; as soon as steam rises pour into a watch-glass, and place the cover-glass upon the stain. Allow it to remain four or five minutes, then wash in methylated spirit until no more colour comes away; drain thoroughly and dry. Mount in Canada balsam.” The bacilli of tubercule are stained red, all other organisms blue.

Echinococcus-vesicles are in rare cases to be found in the

\* *Berl. kl. Woch.*, 10th April, 1882.

† See *Lancet*, 5th May, 1883.

expectoration, having either been previously encysted in the lung, or having bored their way from the liver into a bronchus.

(7.) *Foreign Bodies*.—To the presence of carbonaceous particles in the sputum allusion has already been made. Fragments of food, when present, are easily recognised by means of the microscope.

## CHAPTER XV.

### Respiratory System—(*continued*).

#### EXAMINATION OF NARES AND LARYNX.

WE now proceed to the physical examination of the organs of respiration, and these will be considered in the order in which they naturally come—the Nares, the Pharynx, the Larynx and Trachea, and the Lungs.

##### NARES.

The examination of the nostrils appertains more to the domain of surgery than to that of medicine; but it must be briefly alluded to here. Obstruction of the nasal passages obliges the patient to breathe through the mouth; and the effect of this upon the moisture of the tongue and lips has been already commented upon in Chapter I. The resonance of the nasal cavities is of great importance in connection with the voice; and when this resonance is interfered with by obstruction of the nares, the voice acquires a peculiar and characteristic nasal sound. The nares may be examined by—

(1.) *Palpation*.—The finger may to a certain extent be made to explore both anterior and posterior nares, and the presence of polypi and other tumours may thus be ascertained.

(2.) *Inspection*.—The anterior nares may be inspected with the aid of a nasal speculum. With such an instrument it is often possible to examine the mucous membrane of the nostrils and the posterior wall of the pharynx, and even the orifices of the Eustachian tubes. The more posterior nasal structures are,

however, best brought into view by means of *rhinoscopy*. This method of examination closely resembles laryngoscopy (to be presently described). The mirror is, however, turned in the reverse direction; it is passed over the back of the tongue until it touches the posterior wall of the pharynx, where it is held with the surface directed upwards and forwards. If the uvula hang loosely downwards it presents no impediment; but should it contract, turn backwards, and close up the posterior nares, it must be drawn forwards with a hook. Having in this way obtained a view of the posterior nares of the upper, middle, and lower passages, and of the openings of the Eustachian tubes, we should note the colour of the mucous membrane covering these parts, and the presence or absence of swelling, ulceration, new formations, foreign bodies, &c.

#### PHARYNX.

The pharyngeal passage, forming part of the alimentary tract as well as of the respiratory, has been already described in sufficient detail, and need not again detain us at this point.

#### LARYNX.

**Voice.**—As an index of the state of the larynx, the voice is of the utmost importance. All affections of the vocal cords, whether ulceration, swelling, or new formation, and all acute and subacute inflammations of the larynx, are followed by huskiness of the voice. When, therefore the voice is clear and good, all such affections may be excluded. Aphonia (*α, priv., φωνη, sound*), or loss of voice, may, however, result from other causes, such as paralysis of the muscles of the larynx, which may be due to central or peripheral nerve affection, or simply to the exhaustion of severe disease; or it may be of a purely functional nature as met with in hysteria. A degree of aphonia may occur along with considerable dyspnoea owing to the embarrassment of breathing; and where from central nervous disease the respiratory muscles are paralysed, the voice fails.

Aphonia must, of course, be distinguished from aphasia (loss of speech), in which the power of phonation is not affected, and also from deaf-mutism.

**Laryngeal Palpation.**—External palpation of the larynx may occasionally detect tender points, the result of inflammatory changes, which may be more clearly localised by laryngoscopic examination. By placing a finger on either side of the larynx whilst the patient speaks, the transmitted vibration of the vocal cords can be clearly felt. Normally this is equal on both sides, but should one cord be paralysed, the vibration will fail on the corresponding side, and in this way a rapid and accurate diagnosis can sometimes be made.

Internal palpation of the larynx—that is, the examination of the rima glottidis and neighbouring parts with the finger, is not so readily performed. The patient when seated in front of the operator must be made to bend the head back, to open wide the mouth, and to thrust out the tongue. The right forefinger of the operator must then be passed rapidly backward along the roof of the mouth, and then suddenly bent downwards until its tip comes in contact with the epiglottis and neighbouring parts. This method of examination is chiefly useful to detect the presence of œdema glottidis, and of tumours lying over the orifice of the larynx, and to remove foreign bodies which may have become lodged there.

**Laryngoscopic Examination.**—It is not necessary here to describe the various forms of laryngoscopes which have been devised, and many of which are now in use by different observers. For general purposes it suffices to have three or four throat mirrors of different sizes, which are plane mirrors, usually round in shape, attached at an angle of about  $120^{\circ}$  to their metallic rods, which fit into an ivory or wooden handle, and are there secured by means of a screw. It is of some importance to reserve one of these mirrors for syphilitic cases. The light used for illumination may be sunlight or daylight, but in

this climate it is better to trust to the artificial light of a good lamp. The rays of light are reflected into the patient's mouth from a concave mirror, which is most usually secured to the forehead of the operator by means of a circular band passing round the head. This mirror has a small opening in the centre, which is placed so as to correspond to the eye of the observer, and through which he can see the laryngeal mirror.

*Method of Examination.*—The patient should be seated opposite and very close to the observer, with his head inclined slightly backwards, and the fauces illuminated from the mirror on the forehead of the latter. The light should be placed near the side of the patient's head, at the level of and slightly behind the mouth. The patient having been made to open his mouth widely, and to protrude his tongue, grasping its point between the thumb and forefinger of his right hand, protected with a handkerchief or napkin, the observer throws a beam of light on to the fauces from the mirror on his forehead. He now takes the laryngeal mirror in the right hand, warms it gently over the lamp (to prevent the moisture of the expired air from condensing on it), and, after testing its temperature on the back of his hand, he introduces it into the patient's mouth. Holding it as one holds a pen, he passes it rapidly to the back of the throat, carefully avoiding unnecessary contact with the surface of the tongue or palate, and presses its posterior surface upon the uvula. It will be found to increase the steadiness of this movement if the third and fourth fingers of the operator be allowed to rest on the patient's cheek at the angle of the mouth. With the mirror lying in this position the larynx usually comes more or less perfectly into view, when the patient is desired to ejaculate "ah." By slight movements of the mirror the whole of the larynx can be explored. Turning the laryngeal mirror downwards, there come into view in succession the back of the tongue, the upper surface of the epiglottis with the glosso-epiglottidean ligaments, the arytenoid cartilages, the false and the true vocal cords, the ventricles of

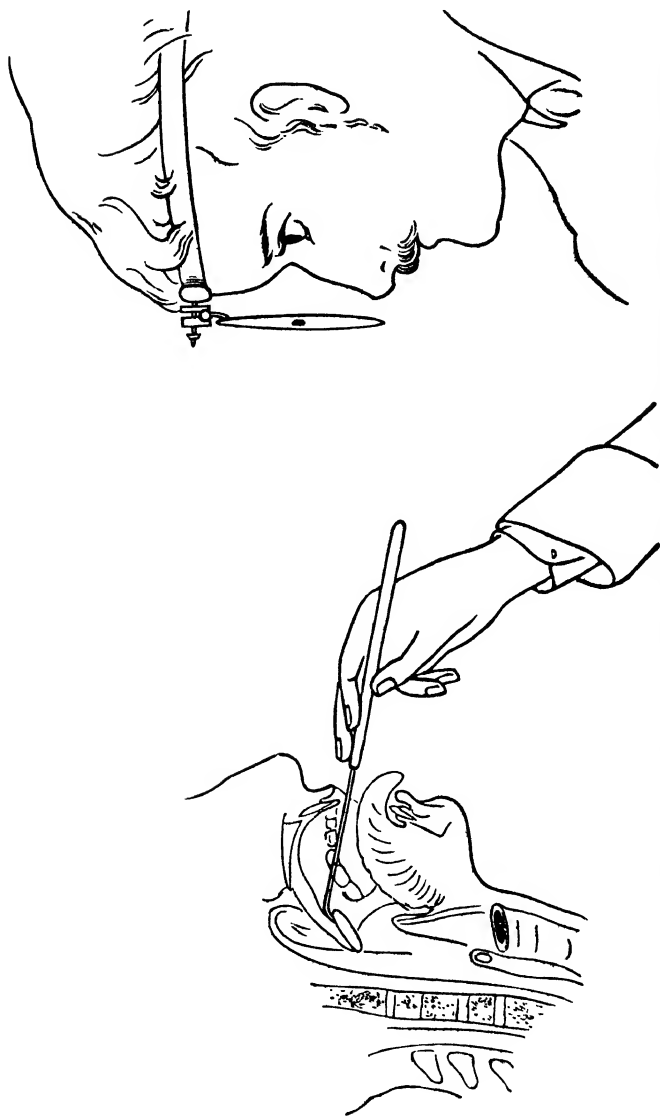


FIG. 19a.—Method of using the Laryngoscope. To show the position of the fingers the hand of the observer has been drawn as occupying a position too far removed from the patient's cheek, on which the fingers ought to rest.



the larynx, the rings of the trachea, sometimes even as far down as the bifurcation.

There are several difficulties which may arise in the course of laryngoscopic examination. Apart from the obstruction occasioned by enlarged tonsils, the observer may have to combat incessant attempts to retch, caused by the contact of the laryngeal mirror with the throat, especially when that region is sensitive. This can best be overcome by allowing the patient to suck small pieces of ice for some little time (twenty minutes) before the examination. In other cases the back part of the tongue may arch upwards to such an extent as to

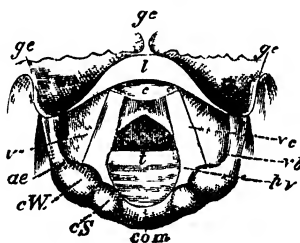


FIG 20 —Diagram of Laryngoscopic Image in Quiet Inspiration (Morell Mackenzie)

- |                                 |                            |
|---------------------------------|----------------------------|
| (ge) Glosso epiglottic folds    | (cs) Capitulum Santorini   |
| (u) Upper surface of epiglottis | (com) Arytenoid commissure |
| (l) Lip of epiglottis           | (vc) Vocal cord            |
| (c) Cushion of epiglottis       | (vb) Ventricular band      |
| (v) Ventricle of larynx         | (vp) Processus vocalis     |
| (ae) Ary-epiglottic fold        | (cr) Cricoid cartilage     |
| (cW) Cartilage of Wrisberg      | (t) Rings of trachea.      |

shut out the view. Very frequently this difficulty disappears when the patient is directed to say "ah;" but if this measure fails, then the organ may be forcibly depressed by means of a spatula. The most troublesome condition of all is met with in those cases in which the epiglottis is so large as to shut out the view of the larynx. Sometimes it can be sufficiently raised by causing the patient to sing a high note, but occasionally this fails. Hooks and other instruments have been designed for the purpose of raising the epiglottis in such circumstances, but their utility is doubtful.

It is to be borne in mind that the parts reflected in the right and left sides of the mirror (in relation to the *observer*) correspond to the *patient's* left and right, and that the more anterior structures are seen in the upper part of the mirror, the more posterior in the lower.

The position of the various structures seen in the laryngeal image is shown in figs. 20 and 21.

Having carefully examined the various portions of the larynx which may be brought into view by changing the position of the mirror, it is perhaps best to systematise the information



FIG. 21.—Diagram of Laryngoscopic Image in the Act of Vocalisation  
(Morell Mackenzie).

- |                             |                             |
|-----------------------------|-----------------------------|
| (fi) Fossa innominata       | (cS.) Capitulum Santorini.  |
| (sp) Sinus pyriformis.      | (a.) Arytenoid cartilages   |
| (ch.) Corner of hyoid bone  | (com) Arytenoid commissure. |
| (cW) Cartilage of Wrisberg. | (pv.) Processus vocalis.    |

gained by grouping the facts under some such heads as the following :—

(1.) *Changes in Colour.*—In the normal larynx the mucous membrane generally has a clear red appearance, while the epiglottis is slightly yellow, and the true vocal cords stand out distinctly, having a clear pearly-white colour. In anæmic conditions, the interior of the larynx generally becomes paler in colour; whereas in acute catarrhal conditions the parts assume an intense red, especially the epiglottis, and even the true cords lose their whiteness and become swollen, red, and injected. In more chronic catarrh the tint assumed is not so

bright, but is more greyish red. In cases of croup the false membrane can sometimes be seen in the larynx.

(2.) *Ulceration*.—If ulcers are visible, their position, size, and general features should be noted.

(3.) *Tumefaction*.—Swelling of the parts round the glottis (*œdema glottidis*) may occur from a variety of causes, and the early recognition of this very dangerous condition by means of the laryngoscope is of the utmost importance. New formations are sometimes met with, and their size, character, and seat must be carefully noted.

(4.) *Foreign Bodies*.—In children, in particular, the laryngoscope is of great service in showing where a foreign body has lodged, and in aiding in its removal.

(5.) *Movements of the Larynx, particularly of the True Cords*.—The larynx is supplied by means of two nerves, the superior and the inferior (or recurrent laryngeal branches of the vagus). Paralysis of these nerves produces different symptoms, and must be considered separately. The movements of the cords are very readily discerned. During inspiration they move apart, so as to leave a very free passage for the air, while during expiration near each other again. These movements are, of course, exaggerated during forced breathing. When a note is sung the vocal cords of each side approach very close to each other, so as almost to come in contact. The position of the cords in inspiration and phonation respectively is shown in figs. 20 and 21.

**Paralysis of the Superior Laryngeal Nerve** causes anæsthesia\* of the mucous membrane of the upper and middle portions of the laryngeal cavity, along with paralysis of the cricothyroid muscle, and of the depressors of the epiglottis (thyro and aryteno-epiglottidean muscles). As a consequence of this

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\* In making a diagnosis of anæsthesia of the larynx the sensibility to touch can be tested by means of a probe, and that to pain by means of a fine electrode, both instruments being manipulated with the guidance afforded by the laryngeal mirror.

the epiglottis is immoveable, and is applied to the back of the tongue ; and as without the action of the crico-thyroid the vocal cords cannot be rendered tense, the voice is hoarse and deep, and (according to Mackenzie) the cords can be seen to be loose, their centre being visibly depressed during inspiration, and elevated during expiration. The paralysis of the epiglottis allows particles of food to enter the larynx. Paralysis of the superior laryngeal nerve is usually the result of diphtheria.

**Paralysis of the Inferior (or Recurrent) Laryngeal Nerve** may be due to central degenerative changes in the floor of the fourth ventricle (in bulbar paralysis, and other affections of the pons and medulla), but it is much more frequently the result of lesions of the nerve trunks in their course. In rare instances this paralysis is due to injury of the pneumogastric nerve such as that arising from the pressure of tumours ; much more commonly the nerve trunk pressed upon is that of the recurrent branch itself. Whilst the two recurrent nerves are equally liable to suffer from the pressure of such tumours as bronchocele, cancer of the upper part of the œsophagus, &c., it is to be carefully borne in mind that the course of the left nerve round the aorta exposes it specially to injury from the pressure of aortic aneurisms, while the right recurrent is frequently paralysed by being involved in thickening of the right pleura with which it lies in contact—a condition met with in phthisis of the right apex.



FIG. 22.—Unilateral recurrent Paralysis—phonation (after Ziemssen).

**Bilateral Recurrent Paralysis.**—In this very rare condition the vocal cords are perfectly immobile, and may be seen to have assumed what Von Ziemssen calls the cadaveric position, that, namely, which is found in the dead subject ;\* there is

\* The cords lie about midway between the lateral position of deep inspiration and the median one of phonation.

absolute loss of voice, and the patient speaks in a whisper, and that with considerable exertion and difficulty, owing to the great expenditure of air required on account of the width of the glottis. For the same reason coughing becomes extremely difficult, but there is little or no dyspnoea.



FIG. 23.—Unilateral recurrent Paralysis—inspiration (after Ziemssen).

*Unilateral Recurrent Paralysis.*—In this condition the vocal cord on the paralysed side occupies the cadaveric position already described, while the healthy cord possesses its normal range of movement, and, indeed, rather exceeds this, crossing the median line to some extent so as to compensate for its paralysed neighbour. The voice is impure, metallic, and high pitched, and readily, as Traube pointed out, passes into a falsetto.

### **Paralysis of the Individual Muscles supplied by the Inferior Laryngeal Nerve.**

(1.) *Posterior Crico-Arytenoid Muscles.*—These muscles have for their function the widening of the glottis, which is necessary for inspiration. When they are both paralysed, a condition ensues which is one of the gravest met with in connection with laryngeal pathology. The two vocal cords are then found to be lying close to each other in the middle line, and from this position they do not move even during inspiration. The consequence is, that when the paralysis is complete there is very well marked inspiratory dyspnoea, and this not merely because the vocal cords cannot be drawn asunder by the paralysed muscles, but because the rush of air forces them still more closely together. While inspiration is, therefore, noisy, the voice is usually unaffected.

When only one of the posterior crico-arytenoid muscles is paralysed, the vocal cord of the affected side lies in the middle

line; the voice is impure, but the glottis being comparatively wide, it is only with forced inspiration that there is any noise.

(2.) *Arytenoid Muscle*.—This muscle having for its function the closure of the posterior third of the glottis, it will be easily understood that when it is paralysed both vocal cords lie during phonation in their normal position for the anterior two-thirds of their length, while at the posterior end of the glottis an open triangle is left through which air escapes unhindered. As a consequence of this the voice is hoarse and impure.

(3.) *Internal Thyro-Arytenoid Muscles*.—The action of these muscles is to render the vocal cords tense, and thereby to close the glottis. When one is paralysed, the cord of the corresponding side is lax, and shows a slight concave excavation of its inner edge. When the paralysis is bilateral, this excavation is of course found in both cords; the voice becomes hoarse, and speaking difficult.

The symptoms of individual paralysis of other muscles of the larynx have not yet been clearly ascertained.



FIG. 24.—Paralysis of Arytenoid Muscle (Morell Mackenzie).



FIG. 25.—Paralysis of internal Thyro arytenoid Muscles (Morell Mackenzie).

## CHAPTER XVI.

### Respiratory System—(continued).

#### INSPECTION.

IN order to determine the position of any particular point on the thoracic wall for the purpose of description or record, the thorax has been divided arbitrarily into certain regions, which may be grouped in the following manner:—

1. *Median or sternal group*, bounded on either side by the sternal border, which comprises—

- (a.) Supra-sternal notch.
- (b.) Superior sternal region.
- (c.) Inferior sternal region. The two last regions are separated by a horizontal line corresponding to the level of the lower border of the third costal cartilage.

2. *Antero-lateral group*, bounded internally by the sternal border, and externally by a line which commences at the first ring of the trachea, runs diagonally outward to the acromion process, and then falls vertically downwards. This group comprises—

- (a.) Supra-clavicular region, lying above the upper edge of the clavicle.
- (b.) Clavicular region, corresponding to the inner half of the clavicle.
- (c.) Infra-clavicular region, from the clavicle to the lower border of the third rib.

- (d.) Mammary region, from the third to the sixth rib.
- (e.) Infra-mammary region, from the sixth rib downwards.

3. *The lateral group* corresponds to the axilla, being bounded anteriorly by the vertical acromial line, which limits the anterolateral group, and posteriorly by the axillary border of scapula. This group comprises—

- (a.) Axillary region.
- (b.) Infra-axillary region, which is separated from the former by a horizontal line at the level of the sixth rib.

4. *Posterior group*, bounded externally by the axillary border of the scapula, and internally by the middle line posteriorly. The members of this group are—

- (a.) Supra-scapular region, lying above the scapula.
- (b.) Supra-spinous region, corresponding to the supra-spinous fossa.
- (c.) Infra-spinous region, corresponding to the infra-spinous fossa.
- (d.) Infra-scapular region, lying below the scapula.
- (e.) Inter-scapular region, lying between the scapula and the middle line.

#### INSPECTION.

For the inspection of the thorax the patient should be placed in a good light, if possible in a sitting posture, in an unconstrained position, and with the surface of the chest fully exposed. The general outline of the thorax ought to be viewed from the front, from the back, from either side, and from above and behind, looking downwards. Such inspection gives information concerning (1) the form, and (2) the movements of the chest.

**1. The Form of the Chest.**—The typical chest formation, which is, however, but rarely met with, may be said to possess



the following characteristics. Conical in form, with the antero-posterior diameter shorter than the transverse, it is symmetrical on both sides, both generally and at each corresponding point. The supra- and infra-clavicular regions are almost on a level with the clavicles, and from the collar-bones downwards to the fourth rib there is on either side a gentle convexity. The nipple is placed (in the male and virgin female) on the fourth

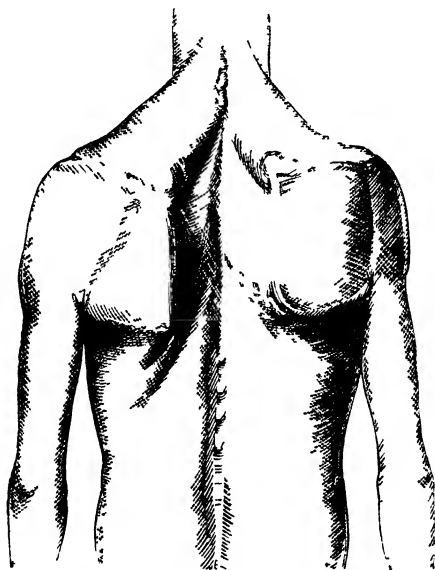


FIG 26 — Alar Chest (R Thompson)

rib or fourth intercostal space, and from this point downwards the chest wall becomes somewhat flattened. In the upper two-thirds of the chest the outlines of the ribs are not well defined, but below this the thinner covering of muscle allows their form to become apparent. The spine and sternum occupy an almost exactly median position, and the shoulder-blades are symmetrical.

From this typical form there are many deviations compatible

with health (physiological heteromorphisms, as Woillez \* terms them), of which the principal are those associated with the phthisical and with the rickety constitutions. Many persons who are predisposed to phthisis show a peculiar thoracic conformation which has been called alar, or pterygoid, on account of the wing-like projection of the scapulæ. The chest is long, narrow, sometimes flattened anteriorly, the ribs oblique, the shoulders sloping, and the throat prominent.

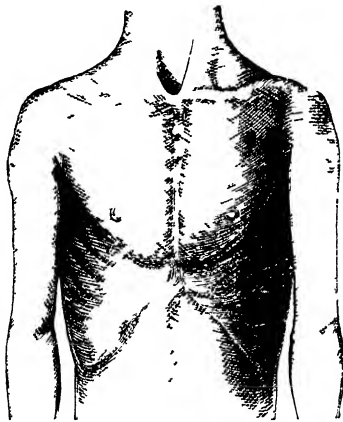


FIG 27 —Pigeon Breast (R Thompson )

The occurrence of any obstruction to the respiration in childhood along with rickets tends to produce the “pigeon-breast,” through the yielding of the softened ribs. In this form of thorax the ribs are straightened, and the sternum thrown forwards so that a transverse section of the chest would approach a triangular form. Independently, however, of any pulmonary complication, rickets may of itself determine a peculiar thoracic formation, when the ribs are so soft as not to be capable of bearing the atmospheric pressure necessarily thrown upon them

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\* *Traité de Percussion*, &c., p 415. Paris, 1879.

during inspiration. A longitudinal groove is thus formed on either side of the sternum.

Irregular formation of the thorax may also be caused by deformities of the spinal column.

*Changes in the Form of the Thorax in Pulmonary Diseases.*

These may be local or general.

(1.) *Local.*—Bulging is met with in encapsuled pleural effusions, in empyæma, in pericardial effusions, in cardiac hyper-

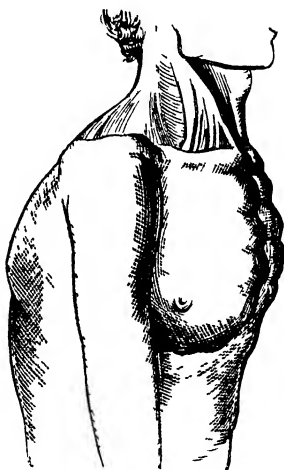


FIG 28 — Emphysematous Chest (R Thompson)

trophy, and over large cavities in the lung. Tumours of the liver and spleen may also cause bulging, the former at the right side, the latter at the left, and surgical affections of the chest wall may give rise to local swelling. Localised shrinking occurs chiefly in connection with phthisis, when there may be flattening in the supra- and infra-clavicular regions. The rare condition in which there is congenital absence of part of the pectoral muscles must not be mistaken for flattening.

(2.) *General*.—Bilateral enlargement of the thorax results from pulmonary emphysema. This so-called barrel-shaped chest is enlarged in all its diameters, rounded, and the intercostal spaces wide. The respiratory movements are very slight, and the thorax remains permanently in a condition resembling that of full inspiration. Unilateral enlargement may arise from extensive pneumonia, from tumour affecting the greater part of one lung (Eichhorst), but it is most evident when effusion of fluid or gas takes place into the pleural cavity. In pleurisy with extensive effusion the diameter of the thorax on the affected side is increased; the intercostal spaces are wide, and rise to the level of the ribs, or even bulge beyond them; the nipple is moved upwards and outwards, and the heart is pressed over towards the sound side in the manner already described. Unilateral shrinking of the chest may come on as the result of absorption of a pleural effusion when the lung is not in a condition to expand. It is also met with in cases of pulmonary cirrhosis.

**2. Respiratory Movement.**—In connection with the act of breathing we have to note the following points—(1) its frequency, (2) its rhythm, (3) its type, (4) its pain or difficulty, (5) the extent of the movements.

(1.) *The Frequency of Respiration*.—The respiratory movements are so much under the control of the will that the physician should endeavour to estimate their rapidity without the knowledge of the patient. This is best done by holding the fingers upon the radial artery, as if to count its pulsations, while the patient's hand rests upon the epigastrium and rises and falls with the respiration. Whilst in new-born children the breathing may be at the rate of forty-four per minute, in the adult male it averages from sixteen to twenty-four, but is slightly more rapid in the female. It is increased in rapidity by exertion, and after meals, and is lower in the recumbent posture than when sitting or standing. It reaches its lowest rate during sleep. It is most important to note the ratio

between respiration and pulse, which is usually 1 : 4, but many vary from 1 : 1 to 1 : 7.

Pathologically, the act of breathing is rendered slow by stenosis of the larynx (as in croup), and by any cerebral disease which interferes with the respiratory centre in the medulla. More common, however, is increase in frequency, which may arise in a variety of ways—(1) When the act of respiration causes pain (as in pleurisy and peritonitis); (2) reflexly, as a result of pain situated in other organs; (3) from chemical changes in the blood, such as are met with in anæmia and in leucocythæmia; (4) from mechanical hindrance to the entrance of air into the pulmonary air cells, which may exist in the larynx, trachea, bronchi, or lung tissue; or may result from pressure on the lung from without by means of pleural effusion, ascites, meteorism, &c.; (5) disturbance of the circulation through the lungs, which may be caused by a variety of conditions, such as valvular disease, pulmonary embolism, &c.; (6) from fever (the increased temperature of the blood over-exciting the respiratory centre—the so-called “heat dyspnœa”); (7) from certain nervous disorders, such as hysteria.

(2.) *The Rhythm of the Respiratory Movements.*—In health the rhythm of the breathing, when uninfluenced by will, is very regular, expiration following inspiration immediately, and being somewhat shorter in duration, after which there is a short pause. Walshe\* calculates that if the whole act be taken as equal to 10, then the inspiration may be estimated as 5, the expiration as 4, and the pause as 1. These relations, however, only hold good in health. In disease either the expiration or the inspiration may be altered in duration usually at the expense of the pause. Inspiration is lengthened whenever an obstacle to the entrance of air exists in the larynx or trachea, and is particularly well marked in case of paralysis of the posterior crico-arytenoid muscles. Expiration, on the other

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\* “Diseases of the Lungs,” 4th edition, p. 14.

hand, is prolonged when any obstruction to the exit of air exists in any part of the respiratory tract, as is the case in vesicular emphysema. The rhythm of the respiratory movements frequently becomes jerking and unequal, particularly in children, where the flexible chest wall yields to the external atmospheric pressure during inspiration, when any obstruction to the free entrance of air exists in the larynx, trachea, or bronchi.

One of the most peculiar alterations in rhythm is seen in the Cheyne-Stokes breathing, when the sequence of the recurring respiratory acts is broken by the occurrence, at intervals of about 1 to  $1\frac{1}{2}$  minute, of pauses, during which respiration entirely ceases. These pauses, which last from  $\frac{1}{4}$  to  $\frac{3}{4}$  of a minute, are followed by the gradual resumption of the respirations, which, at first short and superficial, grow gradually deeper up to the point of dyspnœa, after which the breathing becomes again shallower until the next pause is reached, and so on. The exact manner in which this peculiar rhythm is produced is somewhat doubtful. One most important factor seems to be a deficient supply of oxygen to the respiratory centre in the medulla. The Cheyne-Stokes breathing is met with in many cerebral diseases, in uræmia, in fatty degeneration, in valvular disease of the heart, and is usually one of the immediate precursors of a fatal termination.

(3.) *The Type of the Respiratory Movements.*—In men the respiratory movements chiefly affect the abdominal walls and the lower ribs (costo-abdominal type), while in women the diaphragm does not take so prominent a part in the act of breathing, and the movement is in great measure confined to the upper part of the thorax (costal type). In disease, however, these conditions may be changed, for anything which interferes with the movements of the diaphragm (such as ascites, peritonitis, and many other affections of the abdomen) will in a man change the type of breathing into the purely costal; whilst the latter type of breathing may be lost in a woman when there is some painful affection of the thoracic organs which obliges the respiration to be chiefly abdominal.

(4.) *Pain and Difficulty in Breathing.*—Pain in relation to the organs of respiration has already been mentioned, and need not detain us here. When present, it is usually, though not always, aggravated by the respiratory movements.

*Dyspnœa*, or difficulty in breathing, may be of a purely nervous or subjective kind. The rate is then usually normal, and the air can be heard to be freely entering the lung, while all the causes of objective dyspnœa are absent. This symptom usually occurs in hysterical women. In true objective dyspnœa the respirations are deep and long drawn, and, as already mentioned, either the inspiration or the expiration may be interfered with. In the former case all the accessory muscles of inspiration are called into play, and their contractions form a very striking feature in such cases. These include not merely the dilators of the thorax, such as the sternomastoids, the scaleni, the pectorals, &c., but also those muscles which dilate the nostrils, elevate the soft palate, depress the larynx, and open the glottis. In order to the effective action of these accessory muscles, the patient has to assume the sitting posture (*orthopnœa*), and, provided that he is not comatose, the degree of difficulty of breathing which is present may be more or less accurately estimated, according as the position assumed approaches the sitting posture.

(5.) *The Extent of the Movements.*—In the barrel-shaped chest which accompanies vesicular emphysema, there is, as has been already said, diminution of the movements of the chest in all directions. More important, however, for the purposes of diagnosis, are those localised inequalities in the range of the movements which are occasionally met with. When one lung is compressed by reason of pleuritic effusion, or is from any other cause rendered incapable of expansion, the thoracic movements on that side become defective. Phthisical consolidation at the apices gives rise to deficient movement in the upper part of the chest, as compared with the lower; while in cases of stenosis of the larynx and in emphysema, the opposite

condition obtains, for then during the expansion of the chest there is depression of the lower intercostal spaces, of the epigastrium, in the supra-clavicular regions, and in the supra-sternal notch.



## CHAPTER XVII.

### **Respiratory System—***(continued).*

#### PALPATION.

By laying the hand flat upon the thorax and palpating its walls information may be obtained regarding the form and movements of the chest, the presence or absence of fremitus, of fluctuation, and of certain pulsatory movements other than those already referred to in connection with the circulatory system.

**1. The Form of the Thorax.**—The general form of the chest is best appreciated by means of simple inspection ; but localised changes in shape may be recognised by palpation.

**2. The Movements of the Thorax.**—The information obtained by inspection may be supplemented by laying the hands on the thorax, and estimating the local movements of expansion and elevation at particular parts.

**3. Vocal Fremitus,** or that vibration of the chest wall which may be felt in a healthy person while speaking, is of considerable diagnostic importance. Under the vocal cords in the larynx lies an air column, which extends through the trachea and bronchi to the pulmonary alveoli, and which is set in vibration when the vocal cords vibrate, and through the bronchial walls and lung tissue the thrill so generated is conducted to the thoracic parietes. It is not difficult to understand the conditions under which its intensity becomes increased

or diminished. The thickness of the thoracic wall has an important influence, the thrill being more distinct in emaciated subjects than in those who have much deposit of fat underneath the skin. The intensity of the vocal fremitus also depends upon the loudness of the tone spoken, and upon the depth of its pitch; and finally, it must not be forgotten that it is more distinct in men than in women and children, and that the thrill on the right side is almost invariably greater than that on the left; this being accounted for by the larger calibre of the right bronchus.

In disease the vocal fremitus may be diminished or increased.

(1.) *Diminished*.—Any condition which blocks up the bronchi, such as collection of mucus or pus, or compression by means of tumours, will produce a diminution of the vocal fremitus over the corresponding part of the chest wall. More extensive loss of the thrill is met with where effusion of fluid or gas into the pleural cavity has taken place. If the effusion be extensive, the vocal fremitus may be entirely lost; and should the lung be bound down by extensive adhesions, the fremitus may not be regained even after the entire absorption of the effusion.

(2.) *Increased*.—When infiltration takes place into the air-cells of the lung, the pulmonary parenchyma becomes at once a better conductor of the vocal vibrations, and in consequence the thrill becomes intensified. Such is the case, for example, in lobar pneumonia; and where the lower lobe is affected, the vocal fremitus gives most important aid in distinguishing that affection from pleural effusion. The fremitus is likewise increased where there is phthisical consolidation, particularly if cavities have formed; but should a main bronchial branch leading to the part have become obstructed, either by pressure or by the collection of mucus, pus, or blood, the vocal thrill may be diminished or lost.

**4. Pleural, Bronchial, and Cavernous Thrills.**—The palpitating hand may also detect the fremitus occasioned by the

rubbing together of the roughened pleural surfaces in cases of pleurisy, and large rales in the bronchi or in cavities in the lungs may communicate an appreciable thrill to the walls of the chest. These are, however, of little diagnostic importance, in that they are better appreciated by the aid of auscultation.

5. **Fluctuation.**—When one side of the thorax is distended with fluid, fluctuation may occasionally be detected in it, more particularly and importantly in empyæma.

**Aspiration.**—The general rules for the use of the aspirator have already been given with sufficient fulness (see p. 65), and it is only necessary now to add that in cases of pleural effusion, where the diagnosis is in any respect doubtful, the use of the aspirator is indicated. For this purpose a hypodermic syringe is the most handy instrument. The needle should be introduced through an intercostal space, just above a rib, preferably in the line of the angle of the scapula, and, of course, below the level of percussion dulness.

## CHAPTER XVIII.

### Respiratory System—(*continued*).

#### MENSURATION.

MENSURATION, which is intended to render precise the information which may be gained by inspection and palpation, and which in some of its developments passes much beyond these, is performed by the aid of a variety of instruments which fall to be described in detail.

1. **The Tape-Measure** is used to ascertain the circumference of the chest, which, at the level of the nipples and at the end of expiration, measures in the healthy male adult about thirty-two or thirty-three inches. A full inspiration increases this figure by from two to five inches, while in quiet respiration the inspiration position exceeds the expiration by about half-an-inch. Unfortunately the circumferential measurement of the chest is of comparatively little diagnostic value, as very great variations are met with in health. Much more important is it to learn the relative size of the two sides of the chest. This is most conveniently done by joining two tapes at the commencement of their scales, and fixing this point of junction over the vertebral column. Each side of the chest has thus a tape for itself, and the two measurements can be simultaneously made and compared. In right-handed persons the right half of the chest is about half-an-inch larger in circumference than the left; while in those who are left-handed these conditions are either reversed, or,

what is more common, the two sides of the chest are practically identical in size. Unilateral enlargement and shrinking, the result of disease,\* are very readily detected by means of such measurement.

**2. Callipers.**—Various diameters of the chest may be measured by means of a pair of common steel callipers. Of these the most important is undoubtedly the antero-posterior (sterno-vertebral), which in the phthioid chest is much diminished—the normal measurement being 9 to 10 inches. It is more difficult to obtain exact measurements of the antero-posterior diameter of either apex. For this purpose, one point of the callipers is to be applied immediately below the centre of the clavicle, and the other on the spine of the scapula at a similar distance from the middle line. If great care be taken, sufficiently reliable results may in this way be obtained, when it will be found almost invariably, that in healthy persons the right measurement very slightly exceeds the left. An excess of even a fourth of an inch on the right side indicates, however, morbid depression on the left; while, if the left be in excess by that amount, there is still more conclusive evidence of contraction on the right side.†

**3. Cyrtometer.**—This instrument, devised by Woillez, consists of a series of small pieces of whale-bone, so articulated together as to form a stiff chain which, when closely applied to the walls of the thorax, retains the curves given to it, and which, when removed and laid upon a large sheet of paper, permits of these curves being marked out on the paper. The instrument may be more simply constructed of two pieces of lead wire, joined together by means of a piece of india-rubber tubing, and in this form it is very easily used, and the outline of the chest wall can be most accurately depicted by its means.

\* *Vide* page 182.

† Walshe, "Diseases of the Lungs," 4th ed., p. 33.

So far I have described those instruments which are fitted to give us measurements of the chest when at rest, and I now proceed to consider those which have for their design the measurement or registration of the thoracic movements.

4. **Thoracometer, or Chest Measurer.**—This instrument, as constructed by Sibson,\* consists, in its essential parts, of a dial which measures accurately the vertical movements of a small rod, which is applied to the surface of the chest by means of a spring. Owing to various errors which are necessarily present in the readings obtained, this instrument has never come into general use.

5. **Stethograph.**—The double stethograph of Riegel appears to be more trustworthy in its results than the last-named instrument. Two levers, which are acted upon by the movements of the chest walls at two different points, are arranged so as to record their results on a strip of paper, travelling horizontally by clockwork. The tracings so obtained enable us to analyse the respiratory movements in a much more exact manner than can be done by means of any other instrument. Pathologically, the most striking changes are those in which there is an impediment to the free entrance or exit of air. For example, where the larynx or trachea is stenosed, while the expiratory curve is normal the inspiratory is much prolonged. The reverse is the case in emphysema, where the expiratory curve is prolonged and irregular.

I now come to the third class of instruments of mensuration, those, namely, which deal with the air passing into and out of the chest.

6. **Spirometer.**—Hutchinson's spirometer consists of a gasometer properly poised and adjusted, into which the patient expires forcibly through an elastic tube, and which is arranged so as to measure the amount of expired air. The "vital capacity" varies with age, stature, and sex, but when allowances

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\* *Medico-Chirurgical Transactions*, 1858.

have been made, it may be said, as a general rule, that a diminished quantity of air is expired where there is stenosis of larynx, trachea, or bronchi, interference with the free movement of the thoracic walls, or diminution of the respiratory surface of the lungs. Of these diseases the most striking in its results is undoubtedly phthisis.

Hutchinson gives the following table of the results he obtained from very numerous observations:—

STATURE.		Capacity of Healthy Males.	Early Stage of Consumption.	Advanced Stage of Consumption.
		Cubic Inches.	Cubic Inches.	Cubic Inches.
From 5 feet	to 5 feet 1 inch,	174	117	82
" 5 "	1 inch to 5 " 2 "	182	122	86
" 5 "	2 " to 5 " 3 "	190	127	89
" 5 "	3 " to 5 " 4 "	198	133	93
" 5 "	4 " to 5 " 5 "	206	138	97
" 5 "	5 " to 5 " 6 "	214	143	100
" 5 "	6 " to 5 " 7 "	222	149	104
" 5 "	7 " to 5 " 8 "	230	154	108
" 5 "	8 " to 5 " 9 "	238	159	112
" 5 "	9 " to 5 " 10 "	246	165	116
" 5 "	10 " to 5 " 11 "	254	170	119
" 5 "	11 " to 6 "	262	176	123

It must, however, be borne in mind that there are many fallacies in the use of this instrument. Some persons cannot be made to understand how to blow, others by taking great pains attain to higher figures than their average, and finally, by practice, the art of blowing is so readily learned that those accustomed to the instrument can raise the gasometer cylinder to very considerable elevations.

**7. Pneumatometer.**—This instrument, by means of which the force of expiration and inspiration is measured, is most conveniently used in the form devised by Waldenburg, which consists of a simple mercurial manometer connected by means of an elastic tube with a mouthpiece that fits accurately and tightly over the mouth and nose of the patient. When the

patient expires through the tube, the column of mercury sinks in the proximal limb of the manometer and rises in the distal, while with inspiration these movements are of course reversed, and in either case the amount of displacement is to be read off on the scale.\* In a moderately well-developed male the inspiratory pressure is from 70 to 100 mm. of mercury, while the expiratory is from 90 to 130 mm., whereas in the female we get with inspiration a pressure of 50 to 80 mm. and with expiration 70 to 110 mm. Of more importance than the absolute pressure (which varies much in different individuals) is the difference between the expiratory and the inspiratory pressure, and it must be carefully borne in mind by those who use this very valuable instrument, that in healthy persons the power of expiration exceeds that of inspiration by 20 to 30 mm. It is by comparing these two pressures that the most important indications are obtainable. Their relation is altered in disease as follows:—

*Expiratory pressure is increased* in relation to inspiratory in phthisis (even at a very early stage), in stenosis of the air passages, in pulmonary congestion, pneumonia, and pleurisy, and in such abdominal affections as impede respiration by pressing the diaphragm upwards.

*Expiratory pressure is diminished* until it becomes equal to or below the inspiratory in pulmonary emphysema.

Undoubtedly, the importance of the diagnostic indications given by the pneumatometer in cases of incipient phthisis is very considerable, and in all chronic chest complaints the instrument is of value in indicating the progress of the disease—progressive or retrogressive, as the case may be—and in estimating the results of treatment.

The space at my disposal does not permit of a description of various other, less important, instruments.

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\* Since the level of the mercury when at rest corresponds in both limbs to the zero of the scale, the reading obtained must, of course, be doubled to represent the true difference in the level of the two columns.



## CHAPTER XIX.

### Respiratory System—(*continued*).

#### THEORY OF PERCUSSION.

WHEN the surface of the chest is percussed in the manner which will be described in the next chapter, a sound is produced which is called the percussion note of the part. This term *note* is, however, apt to mislead, for it is not a simple or pure note, not being composed of a regular series of simple vibrations, nor is it (as is the case with the sounds produced by musical instruments) made up of one well marked basal or fundamental tone and a series of higher pitched upper partial tones which bear a definite relation to the basal or prime tone. The sound which is heard on percussion of the healthy chest is composed of a large number of tones, bearing no definite relation to one another, and in it no definite or well marked fundamental note can be distinguished. It is very often assumed that the note produced on percussing the front of the chest—for example, at the level of the second rib—is made up of a distinguishable fundamental or prime tone, corresponding in pitch to the antero-posterior diameter at the particular point in question, just as when one blows across the mouth of a test-tube the prime tone obtained corresponds in pitch to the length of the air column contained in the tube. This view is, however, clearly untenable; for, apart from the inherent improbability of this particular air column alone being set in audible vibration, and not the many others which radiate

from the point of percussion to the other limits of the thoracic cavity, there is the fact that in practice it is impossible to find what is the real pitch of this fundamental or prime tone; and, further, it is constantly noticed that the apparent pitch of the percussion note varies enormously with the variety of pleximeter employed, and still more when the pleximeter and finger are compared, which would not be the case were there a distinguishable prime tone.

The percussion note is made up of vibrations which are derived from three sources.

1. *The Vibrations of the Pleximeter.*—When the finger is employed as a pleximeter, these vibrations are practically inaudible. In the case of an ivory pleximeter, however, they are readily recognised. If the instrument be of the usual form, the vibrations are clear and relatively high in pitch; but provided that the pleximeter be properly damped by being firmly pressed upon the thoracic wall, and be struck with the pulp of the finger alone, or with the india-rubber of a hammer, the tone it gives can be readily discounted by the physician.

2. *The Vibrations of the Thoracic Wall.*—These are of so ill-marked a character (unless the point struck lie over the rib of a very thin subject) and have so little intensity as compared with the intrathoracic note, that in themselves they need hardly be considered, though, as I will presently point out, the condition of the chest wall and its vibrations when percussed have a very important influence on the character of the intrathoracic note.

3. *The Vibrations of the Air in the Lungs.*—These vibrations constitute the important part of the percussion note, and must be considered in some detail.

When percussion is made at any point of the chest wall, the air in the lungs is set in vibration, and the point which is struck may be considered as the point of divergence of a series of radiating air columns whose lengths may be represented by lines drawn from the corresponding point on the visceral pleura to the opposite walls of the thorax in all directions. The lengths of

these very numerous columns, of course, differ considerably ; and since an air column, when set in vibration, produces a note proportionate in pitch to the length of the column, the numerous notes which go to make up the percussion sound vary considerably in pitch. The pulmonic septa also, in all probability, limit the length of certain of these air columns, and in others they may determine nodal points, and in this way cause still greater differences in pitch. We have thus to consider the intrathoracic percussion sound as composed of a large number of prime or fundamental tones, which vary much in pitch, and each have an ascending series of upper partial tones which tend, of course, still further to render the vibrations of the combined percussion note irregular.

In a musical note we have to recognise three distinct characters—viz., intensity, pitch, and quality ; and in relation to the percussion sound, these must also be considered.

1. *Intensity*.—The intensity of a musical tone depends upon the amplitude of the individual vibrations of which the tone is composed. In the case of the drum, for example, the intensity of the tone depends upon the vigour with which the drum-head is struck. In the same way, the intensity of the percussion-note depends, to a considerable extent, upon the strength of the stroke. But it must be remembered that the percussion note, as I have just said, is composed of a large number of different tones, so that its intensity in any given case depends also upon the number of these tones which are produced by the blow. Thus, when the greater part of a lung is hepatised, the percussion note over the healthy portion loses much of its intensity, because there are fewer air columns which can be set in vibration. The intensity of the note is, therefore, of considerable diagnostic significance.

2. *Pitch*.—The pitch of a simple tone, such as that of a tuning-fork, depends upon the rate of the vibrations of which it is composed. In the case of a musical note, composed of a prime tone and an ascending series of upper partial tones (as, for example, the note of a stretched cord), the term

pitch is understood to mean the pitch of the prime tone. It is thus clear that, in regard to the percussion sound, we cannot, properly speaking, use the term pitch, since it is impossible to detect any fundamental or prime note. It is evidently advisable, however, to retain the term, which is so useful clinically, provided that in using it we carefully keep in mind that we do not refer to the pitch of a basal note, which, as I have said, does not exist, but to the general pitch of the combination of tones which reach the ear. If we take an illustration from the piano, it will easily be seen what is here meant. Suppose that a number of notes at the treble end of the key-board be struck simultaneously with one or two at the bass end, the general impression will be that of a high pitched sound, and *vice versa*; and so also in the case of the percussion sound, if the number of higher tones preponderates greatly over that of the lower tones, the general sound appears to be high in pitch, and *vice versa*.

It has been said that when the chest wall is struck, the underlying air columns are set in vibration. This is due in great measure to the direct transmission of the impulse, but in some degree, at any rate, these vibrations seem to arise by sympathetic resonance. This demands a few words of explanation.

If a series of tuning-forks, of different pitch, be in turn sounded over the mouth of an empty jar, it will probably be found that the series contains one fork to which the air in the jar, so to speak, answers,—which, when it is sounded, throws the air column into sympathetic vibration, so as to reproduce and strengthen its own note. If the air column be measured, it will now be found that its length is exactly one-fourth the length of the sound-wave produced by the fork in question. This reproduction and reinforcement of the tone is termed resonance. In the same way if a compound tone be sounded in the neighbourhood of such an air column it will be set in sympathetic vibration if the sound-wave of the prime or any of the upper partial tones happens to bear the relation to the length of the column which has just been stated. Now, when the chest

wall is percussed it vibrates, as I have already pointed out, and gives rise to a sound which is usually inaudible, and which, in any case, is of little importance. But underlying it there are numerous air columns, certain of which are of a suitable length to be set in sympathetic vibration by certain of the tones of which the sound of the thoracic wall is composed ; so that the quality and general pitch of the intrathoracic note does to a certain extent depend upon the vibrations of the chest wall. The slight difference in pitch of the percussion sound during expiration and inspiration is thus to be explained. When the chest is in the condition of full inspiration its walls are tenser than during expiration, and, therefore, give a higher pitched note when percussed, which note is reproduced and strengthened by the resonance of the intrathoracic air columns, and thus raises the general pitch of the percussion sound.\*

3. *Quality*.—The quality of a musical note depends upon the number and character of its upper partial tones. Enough has been said in the last pages to explain how this applies to the compound percussion sounds, and the various well-marked qualities which are to be met with clinically will be best discussed and explained in the next chapter, when we come to deal with the practical aspects of percussion.

\* The pitch, then, of the percussion sound at any given point depends upon the length of the air columns which are set in vibration, whether that vibration be produced by direct impulse or by sympathetic resonance ; and the general pitch of the compound percussion sound depends upon whether the high or low pitched notes are of larger number or of greater intensity. But, as I have already said, the length of some at least of these columns is determined by limiting pulmonic septa. If the lung tissue become relaxed, these septa no longer limit the length of the air columns, which then extend back to the opposite wall of the chest, and consequently give a lower pitched note. Thus, as a whole, the percussion sound depends for its pitch upon three factors—(1) the tenseness of the chest wall ; (2) the tenseness of the lung tissue ; (3) the length of the underlying air columns.

## CHAPTER XX.

### Respiratory System—(continued).

#### PERCUSSION OF THE CHEST.

IN the preceding chapter I have attempted to explain the theory on which the practice of percussion rests. It is now necessary to consider it in its clinical and practical aspects. And first, of the

**Methods of Percussion.**—There are two varieties of percussion, the immediate and the mediate.

*Immediate percussion*, or that in which the chest wall is struck directly with the finger, was the method originally employed. It is now almost completely discarded, the only exception being the percussion of the clavicles, which may with advantage be struck with the pulp of the forefinger before the percussion of the chest generally is commenced.

*Mediate percussion*, or that variety in which the finger or pleximeter is laid upon the chest wall and receives the stroke, is now almost universally employed.

As a general rule, it is probably best to use one or more of the fingers of the right hand to give the stroke, and to employ the fore or middle finger of the left hand as a pleximeter, applying its palmar surface firmly to that portion of the chest wall which we wish to percuss. For the right hand a percussion hammer may be substituted, and for a pleximeter we may employ an ivory or vulcanite plate of any of the numerous forms which are to be found in the shops of surgical instrument makers.

Whether the stroke be delivered by means of the bent finger or the hammer, it must be given from the wrist alone, and not from the shoulder or elbow, and the fingers or hammer must be raised from the pleximeter the moment the blow has been given, so as to allow of the free vibration of the chest. Skillful percussion with the fingers is very difficult to acquire, and requires long practice; but all students should learn it, for although hammer percussion is much easier, the physician may often be in circumstances when he is compelled to percuss without the aid of that instrument. Finger percussion is also much better suited to give the feeling of resistance which, as will be presently shown, is often of considerable importance.

The patient, if a male, ought to be stripped to the waist, and if a female, only one thin and soft garment ought to be allowed to interpose between the chest and the finger or pleximeter.

It is of great importance that the chest should be percussed symmetrically, corresponding points on both sides being compared with one another, and it is necessary to see that the patient assumes no position of head, limbs, or trunk which will produce unequal muscular tension on either side.

As a rule, percussion need not be very forcible, though when the chest walls are thickened from the deposition of fat, or are dropsical, a strong blow may be necessary in order to produce a sufficiently audible note.

**The Thoracic Percussion Note.**—In the preceding chapter I have indicated the theoretical basis on which I believe the practice of percussion may safely be held to rest; but whatever their theoretical beliefs, most physicians will agree that the percussion sound depends mainly upon three factors—viz., (1) the thickness and tension of the chest wall, (2) the tension of the pulmonic tissue, and (3) the amount and disposition of the underlying air; and that it is to physical changes in these three factors that we must look for the cause of variations in the percussion note.

In the following pages we shall consider firstly, the changes

in the percussion note which occur as regards (*a.*) intensity, (*b.*) pitch, and (*c.*) quality (such as the tympanitic note, cracked-pot sounds, &c.); secondly, the feeling of resistance during percussion; and thirdly, the topographical percussion of the lungs.

**The Intensity of the Percussion Sound.**—As has been already said, the intensity of a simple pendular tone depends upon the amplitude of its vibrations; but in regard to the compound percussion sound, account must also be taken of the number of air columns which are thrown into vibration, and this depends upon the force of the stroke, upon the condition of the chest walls, and upon the volume of underlying air. Remembering that when two parts of the chest are being compared the force of the stroke must in each case be equal, we may limit our attention to the two last factors.

1. *The Condition of the Chest Wall.*—When the thoracic wall is thickened by the deposition of fat, or by the transudation of serum into its interstices, the subjacent air is thrown with greater difficulty into vibration by the percussion stroke, and the resulting sound is deadened in passing through the thickened chest wall to reach the ear of the physician. The same diminution of the intensity of the sound occurs in health over those portions of the chest which are covered with thick muscle—for example, the scapular regions, and over the pectoralis major; and it must be borne in mind that in labourers in whom, from their occupation, the right pectoralis is considerably more developed than the left, the percussion note is less intense on the right side over that muscle than at a corresponding point on the left. Pleural effusions, also, have the same effect on the intensity of the note, as the layer of fluid prevents the free transmission of the percussion stroke. Such collections of fluid have, of course, a further influence on the note from the compression of the lung tissue which they occasion. The thickening of the pleuræ, which remains after an attack of pleurisy, tends to diminish the intensity of the percussion note, partly owing to the increased thickness of



the chest wall thereby produced, but also, I think, from the manner in which the strong adhesions formed tend to contract, and so bind together the chest wall as seriously to interfere with its free vibration.

2. *The Amount of Air contained in the Chest.*—The intensity of the chest note is diminished whenever, from any cause, there is a serious diminution of the air contained in the chest. This may result either from compression of the lung tissue, so as to expel the air, such as takes place in cases of pleural effusion and of tumours pressing upon the lung, or from infiltration into the alveoli, such as occurs in pulmonary œdema, in the exudative stage of acute pneumonia, and in all the forms of chronic phthisis.

An *increase* in the intensity of the percussion sound is met with (1) where the chest walls are thin, in the young, the old, and in emaciated subjects; and (2) where the volume of air is increased, as is seen when the percussion note of full inspiration is compared with that of expiration; and further, in cases of emphysema, where the absolute volume of intrathoracic air is increased, not merely because many of the pulmonary septa have disappeared and their place has been taken by air, but also on account of the permanent position of the thorax in the condition of full inspiration.

**The Pitch of the Percussion Sound.**—The pitch of a note depends upon the rapidity of the vibrations of which it is composed, and I have explained in the previous chapter how the term pitch may be more or less correctly applied to such a compound sound as that of percussion. The pitch of the thoracic note depends on three factors—(1) the tension of the chest wall, (2) the tension of the lung tissue, and (3) the length of the underlying air columns; and I have already shown how these three conditions tend to modify the pitch of the note.

1. *The State of Tension of the Chest Wall.*—When a full inspiration is made, the tension of the chest wall increases, and

consequently the percussion note tends to rise in pitch, although this tendency is to a certain extent counteracted by the increase of the volume of air in the lungs which then takes place. As a whole, however, the pitch during inspiration is higher than during expiration. In the same way, in pulmonary emphysema the percussion note is usually raised in pitch, owing in great measure to the increased tension of the thorax; and though the intensity of the note (as has been already said) is increased in these cases, I have frequently seen emphysema mistaken by the inexperienced for pulmonary consolidation, owing to the heightened pitch.

2. *The State of Tension of the Pulmonic Tissue.*—The increased tension of the lung tissue, during full inspiration, no doubt tends to heighten the pitch of the percussion note, along with the tension of the chest wall above mentioned. The results of relaxation of the lung tissue will be best described when we come to speak of the tympanitic note.

3. *The Length of the underlying Air Columns.*—Whenever the air-containing cavity lying under the point of percussion becomes more or less filled up, either as the result of effusion of fluid into the pleural cavity, or of effusion or exudation into the alveoli, such as takes place in œdema, pneumonia, and in the various forms of phthisis, the air column becomes shortened, and the percussion note rises in pitch. The same result follows where, from the deposition of new formations in the lung tissue, the air columns become broken up in their length. Within these limits fall the greater number of pathological conditions which are met with in connection with the lungs. In each special case it is not difficult to see how the note becomes modified as regards its pitch.

In like manner is to be explained the change of note which occurs when we pass from the lungs to such solid organs as the liver and heart, and which enables us to map out their outlines in the manner already described. Take the liver for an example: As we percuss downwards in the mamillary line, we reach the upper margin of relative liver dulness—that point,

namely, where the sound first becomes modified. It is here that the liver, lying in the hollow of the diaphragm, first begins to encroach upon the air space, filling it up from behind, and thereby shortening the air columns, and diminishing the volume of underlying air. The intensity of the note is thus diminished, and its pitch rises, and these changes in the percussion note become more and more marked until we come to the upper limit of absolute liver dulness, where no lung tissue interposes itself between the liver and the chest wall, and the note, therefore, becomes absolutely dull. In the same way, the topographical percussion of other solid organs is to be explained.

Passing now to the consideration of certain changes in the quality of the percussion note, we come first, and most importantly to

**The Tympanitic Percussion Note.**—This variety of chest note differs from that of health in that it approaches much more nearly to a pure musical tone—that is, its vibrations become much more regular. The great regularity of these vibrations Gerhardt has shown by means of König's sensitive flame reflected in a rotating mirror. This variety of percussion note is found in perfection over the stomach when that viscus is moderately distended with air. If the stomach be removed from the body, both orifices ligatured, and then moderately distended with air, it will be found to afford a tympanitic note on percussion; but if the distention be continued, a point will be reached when the note loses that peculiar quality and becomes muffled. The reason of this is not far to seek. In the case of moderate distension, the gastric wall is not sufficiently tense to pass into vibration, and thus the sound results simply from the vibrations produced in the contained air; but when the walls become tense from over-distension they also vibrate, and the tones so produced do not harmonise with those of the vibrating air, so that the combined sound is irregular in its vibrations, and therefore no longer tympanitic.

Similarly, if a lung be removed from the body and allowed to collapse, it will, when percussed, give a tympanitic note, which disappears when the lung is again distended with air to a point corresponding to the normal condition. The air in the collapsed lung vibrates as a whole, and the lung tissue is not sufficiently tense to admit either of its passing into vibration, or of the stronger septa breaking up the air columns so as to render the combined note irregular and non-tympanitic, as is the case when the lung is in a state of normal distension. It is to be noted that the pitch of the tympanitic note (which is very readily made out) gives a trustworthy indication of the size of the air cavity, and this is very important as a means of distinguishing the note of the stomach from that of the neighbouring intestines.

Further illustrations of the tympanitic note in health are to be found when percussion is made on the cheek when the mouth is moderately distended with air, or over the trachea. The latter example is of especial value in that it shows another property of the percussion note—viz., that when the orifice of the cavity is narrowed or closed, the pitch of the note falls. If the trachea be percussed, first with the mouth open, and then with it shut, this lowering of the pitch will be readily detected, and it will be still more obvious if the nostrils be at the same time compressed.

From what has just been said, it will be seen that the tympanitic note may occur in the chest under the following pathological conditions:—

1. Relaxation of lung tissue.
2. The presence of underlying air cavities.
3. Pulmonary consolidation, allowing the broncho-tracheal air column to be set in vibration.

1. *Relaxation of Lung Tissue.*—Just as when the lung is removed from the body, and allowed to collapse, it gives a tympanitic note, so when a similar retraction and relaxation of the pulmonic tissue takes place within the thorax, that variety of percussion note may be heard. This is best marked in cases

of pleuritic effusion, which, gravitating to the lower portion of the cavity, floats up the lung and causes retraction of the upper portions. When the effusion is small in amount, this tympanitic note can only be detected over that portion of lung which lies immediately above the upper limit of the fluid, but when the effusion is considerable, the whole upper lobe may be tympanitic on percussion.\* Similarly, effusion into the alveoli (in pneumonia or œdema) may produce a like result. In the first stage of pneumonia the change in the note seems to be produced by relaxation, occasioned by the inflammatory congestion of the lung tissue. Phthisical consolidation of the apices may also be accompanied with an obscurely tympanitic note over the neighbouring portions of lung.

It is particularly to be observed that the pitch of the tympanitic note which occurs under the above conditions is not altered by shutting and opening the mouth.

2. *The Presence of underlying Air Cavities.*—When the pleural cavity becomes filled with air (pneumothorax) a typically tympanitic note results from percussion, provided that the distension be not too great. Its pitch is not altered by opening and closing the mouth. When the cavity contains in addition serum or pus (hydro- or pyo-pneumothorax) the note changes in pitch with the position of the patient, the fluid gravitating to the most dependant part in each instance, and so altering the lengths of the air columns.

Cavities in the lung tissue, when filled with air, of sufficient size, smooth walled, and near to the thoracic wall, also give a tympanitic note, and as they communicate with a bronchus, the pitch of their note varies when the mouth is opened and closed. The position of the long axis of the cavity may also be ascertained, if it contain fluid as well as air, for the movements of the fluid occasioned by alterations in the position of the patient cause changes in the pitch of the note, just as in

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\* When the effusion is excessive, the lung tissue becomes compressed, and the tympanitic quality of the note is consequently lost.

hydro-pneumothorax (Gerhardt). This change is very characteristic of cavities in the lung.

3. *Pulmonary Consolidation, allowing the Broncho-Tracheal Air Column to be set in Vibration.*—I have already alluded to the tracheal sound, which is characteristically tympanitic. In health, however, it is not possible to set in vibration the air column in the bronchi and trachea by percussing over the chest. If, however, the lung tissue be consolidated, the impulse of the percussion stroke may be transmitted to the bronchi, and in this way the tympanitic tracheal note of Williams may be produced. This note is almost always found on the left side—rarely on the right, and it is characteristic of it that the pitch is altered by opening and closing the mouth, but *not* by changes in the position of the patient.

Another note peculiar in quality, which must be mentioned, is the

**Cracked-Pot Sound** (*Bruit de Pot Fêlé*).—The peculiar quality of this sound is caused by the sudden expulsion of air from a cavity through a small opening in its walls, and it is heard when the hands are pressed together and struck upon the knee, in a manner well known to school-boys, so as to produce a noise closely resembling the rattling of coin. It derives the name (which Laennec first gave it) from the resemblance to the sound produced by striking a cracked jar.

The cracked-pot sound occurs under the following conditions:—

1. *In Health.*—In children, when the glottis is narrowed, either during a fit of crying or when a sustained high-pitched note is being sung, percussion of the chest gives this variety of sound, the air being suddenly forced from the lung through the narrow glottis.\*

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\* Also in adults, when the chest is very hairy, and a pleximeter is being used, the cracked-pot sound is apt to be produced. Owing to the instrument not being closely applied to the chest wall, a layer of air intervenes, and a portion is forcibly expelled by the percussion stroke, producing the sound in question. Moistening the hair does away with this difficulty.

2. *In cases of Relaxation of Lung Tissue*, which I have already described as favouring the production of tympanitic percussion, the cracked-pot sound may sometimes be heard.

2. *In cases of Thoracic Fistulæ*, and pneumothorax, when the percussion stroke expels air through the fistula with a hissing sound.

4. *In cases of Pulmonary Excavation*.—This is by far the most frequent cause of the cracked-pot sound, so much so that when consolidation of the lung tissue exists, the cracked-pot sound may be taken as strong evidence of the presence of a cavity.

The last change in quality which we shall here consider is that which is known as

**Amphoric Resonance.**—This sound is similar to one produced by striking on the side of an empty jar or cask, and it owes its peculiar metallic quality to the high-pitched upper partial tones which it possesses, and which are caused by the reflection of the waves of sound from side to side of the closed cavity. These upper partial tones die away slowly.

When speaking of the tympanitic percussion sound, it was pointed out that when the stomach is over-distended with air that quality is lost, the note becoming hard and metallic. Amphoric resonance is then formed, the sound waves being again and again reflected from the tense walls of the viscus.

The conditions necessary for the production of amphoric resonance are that the air-containing cavity should be of considerable size and superficial, that its walls should be smooth and resistant, and that it should either be completely closed or should only communicate with the external air by means of a small opening.

As in the tympanitic note, so also in the amphoric sound, the pitch of the prime tone enables us roughly to estimate the size of the cavity in question.

In thoracic percussion this amphoric echo is met with in two

conditions—over pulmonary cavities, and in pneumothorax. In both these cases it is best to combine auscultation with percussion, the physician listening with a stethoscope in the neighbourhood of the cavity, while an assistant percusses. For percussion it is best to use a pleximeter, and to strike it with some hard substance such as a coin, as the metallic note thus produced brings out by sympathetic resonance the high-pitched upper partials of the cavity.

**Feeling of Resistance during Percussion.**—The sound which the percussion of the chest affords is not the only sensation which is perceived by the physician in consequence of the stroke. There is further a sense of the degree to which the chest walls yield to the force of the blow.

This feeling of resistance may be dependent solely upon such changes in the chest wall as tend to increase its solidity (such as deposit of fat, thickening of the ribs, &c.), but if we except these, it gives a trustworthy and sometimes exceedingly valuable indication of the comparative solidity of underlying organs. Whenever the lung becomes airless, whether from exudation or compression, the resistance is increased; and still more is this the case when effusion of fluid has taken place into the pleura, and most of all over intrathoracic tumours.

Diminution of resistance is met with in cases of pulmonary emphysema, when well marked, and in pneumothorax.

**Topographical and Regional Percussion.**—The limits within which the pulmonary percussion note is heard are of importance, not only in determining the outline of neighbouring solid organs, but as a guide to the physical condition of the lungs themselves.

*The Apices.*—The upper limit of the lung note corresponds to a line which, following at first the clavicular portion of the sternomastoid muscle, curves over to meet the anterior margin of the trapezius, and then passes downwards to the seventh cervical vertebra. This line rises on each side to a point about  $1\frac{1}{4}$



to 2 inches (3 to 5 centimetres, according to Leitz) above the clavicle, being perhaps a trifle higher on the right side. In percussing the apices, care must be taken that the patient's head is not turned to either side, and that the direction as well as the force of the stroke is the same on each side. The shrinking of the apices, both vertically and transversely, is one of the first physical signs of incipient phthisis, and is therefore of considerable importance. In pulmonary emphysema, the limits above given may be overstepped to a considerable extent.

*The anterior margins* approach each other at the level of the second cartilage, being separated only by the anterior mediastinum, and continue downwards parallel to each other as far as the fourth rib, where the margin of the left lung curves outwards to follow the line of the absolute cardiac dulness, as described on page 82; while that of the right lung continues vertically downwards as far as the sixth cartilage, where it joins the inferior margin.

*The inferior margins* are much affected by respiration. Their position during quiet respiration may be taken to be as follows:—

The right lung—

At sternal border,	.	.	.	6th rib.
Parasternal line,	.	.	.	6th rib.
Mammillary line,	.	.	.	7th rib, upper border.
Axillary line,	.	.	.	8th rib.
Scapular line,	.	.	.	9th rib.
At vertebral column,	.	.	.	11th rib.

The left lung—

Axillary line,	.	.	.	8th rib.
Scapular line,	.	.	.	9th rib.
At vertebral column,	.	.	.	11th rib.

With forced respiration the inferior edges of the lungs rise and fall very considerably,—to such an extent, indeed, that in the axillary line there may be a difference of over 3 inches between full expiration and full inspiration. In cases of

emphysema, not only are the lower borders much depressed, but their movement during respiration is greatly interfered with.

The influence of emphysema, and other pathological conditions, on the anterior borders of the lungs, has been already alluded to in connection with the percussion of the heart.

**Regional Percussion.**—The difference of the percussion sound at different parts of the healthy lung depends upon the condition of the chest wall, and upon the number and disposition of the air columns which radiate from the point struck.

*Anteriorly.*—The sound over the apices above the clavicles is clear, but not great in intensity. Below the clavicles the note falls somewhat in pitch, and grows in intensity until we come to the relative dulness of the heart on the left side (lower margin of third rib) and of the liver on the right (fourth interspace, or fifth rib), when in both cases the sound rises in pitch and loses intensity, and does so more and more until the limit of absolute dulness of each solid organ is reached. The right lung is usually slightly duller than the left, owing to the greater development of muscle on the right side. Over the sternum the sound is clear, deep, and resounding, owing in part to the vibrations of that bone, but chiefly to the fact that the air in both lungs is set in vibration.

*Posteriorly.*—In percussing the thorax posteriorly, the patient should be made to cross his arms in front and bend forward. The note over the scapulæ is less clear than that at the lower portions of the back. The lung note can be heard as low down as the tenth or eleventh rib.

*Laterally.*—In the axillary regions the pulmonary note is intense and clear on both sides, until the dulness of the liver is reached on the right side, and that of the spleen on the left.

## CHAPTER XXI.

### Respiratory System—(*continued*).

#### AUSCULTATION.

THE auscultation of the lungs may be performed with the aid of a stethoscope, or more simply by applying the ear to the thoracic wall. For obvious reasons, the former method is the pleasanter both to patient and to physician, and it possesses this further advantage, that, by means of the stethoscope, any abnormal auscultatory phenomenon can be more distinctly localised than is possible if the immediate method be employed. The form of instrument is of comparatively little importance, provided that it fits the ear of the physician. The simple wooden stethoscope answers admirably for all ordinary cases, although sometimes the double instrument of Alison may be made use of with advantage.

The position of the patient is of considerable importance. Where there is a choice, probably the sitting posture is the most convenient, but whatever attitude be adopted it must be unconstrained. Of at least equal moment is the posture of the physician, which should be easy and comfortable. The chest of the patient should, if possible, be fully uncovered; but, failing this, the intervening clothes must be thin, and all friction between them and the stethoscope sedulously avoided. The instrument should be firmly and accurately applied to the chest, and not till then should the physician apply his ear to the upper end, always remembering that the

ear must be moved so as to suit the stethoscope, and not the stethoscope to suit the ear. Attending to these precautions, the whole chest must be carefully examined, corresponding points on the two sides being compared in the same manner as in percussion.

On auscultating the chest there is to be heard at most points a gentle "breezy" sound — the vesicular murmur — which resembles the sighing of wind among leaves, and the special character of which is readily learned by a little practice. It consists of two murmurs, the one corresponding to inspiration and the other to expiration, of which the first is about three times as long as the second, and is softer and higher in pitch.\* Not only do pathological changes in the lungs alter the ordinary respiratory murmur, but they often produce totally different sounds, to which in turn our attention must be directed. In ordinary clinical examination, then, the main points to be attended to in regard to auscultation are—

1. The relative duration of the expiratory and inspiratory murmurs.
2. The character or quality of the breathing sounds.
3. The presence or absence of adventitious sounds of various kinds.
4. The character of the vocal resonance (auscultation of the voice).

**Vesicular Murmur.**—In speaking of murmurs arising in the blood current, I have already pointed out that when a fluid streaming through a tube passes from a narrower into a wider portion, vibrations arise in the fluid owing to the friction of the molecules upon one another, which, if sufficiently rapid, give rise to an audible murmur. This is equally true with regard to gases. Now, in the air passages there are two points at which such an alteration in calibre is to be found—

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\* The expiratory murmur is, however, not unfrequently inaudible in healthy persons.

viz., at the glottis and at the point where the bronchioles enter the alveoli. The rush of air through the narrow glottis sets in vibration the air column contained in the trachea and bronchi, and a blowing murmur results—the tracheal or bronchial murmur—which will be described more fully hereafter; and in a similar manner a murmur arises as the air streams into the air cells. To a combination of these two murmurs, in which the latter predominates, it appears most reasonable to ascribe the formation of the normal vesicular breath sound.\* Whatever be its origin, however, it may be safely held that, when vesicular breathing is heard, the pulmonary alveoli are fulfilling their function, and when it is absent, that that function is in more or less complete abeyance.

The vesicular murmur is to be heard more or less clearly over the whole pulmonary surface, but it varies in distinctness at different parts according to the thickness of the chest wall and the volume of lung tissue underlying the stethoscope. From various causes vesicular breathing may be absent. Thus it may be replaced by bronchial breathing, or it may be inaudible, owing to the loudness of superadded sounds, or to the interposition of a tumour or of fluid between lung and chest wall; or, finally, it may be absent owing to obstruction of a bronchus or to collapse of lung tissue.

The common modifications of vesicular breathing are as follows:—

1. Harsh or puerile.
2. Jerky.
3. Prolongation of the expiratory murmur.
4. Systolic vesicular murmur.

(1.) *Harsh Vesicular Murmur.*—In children the normal

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\* Baas, Gerhardt, and others, however, hold that the vesicular as well as the bronchial murmur arises solely at the glottis, and that the sound is in the former case modified by transmission through the lung tissue. The theory is attractive, but the proof offered seems inadequate, more especially as clinical facts point in the other direction.

vesicular breath sound is clear, sharp, and loud, and this harsh or puerile breathing appears to depend in part upon the thinness of the chest walls and the greater elasticity of the lung tissue.

In adults harsh or puerile breathing usually indicates a catarrhal condition of the bronchial mucous membrane. When heard over the lung tissue generally it is not, as a rule, of so much importance as when the harsh quality is only perceptible at any or both apices, which sign, when persistent, points strongly to incipient phthisis.

(2.) *Jerky Breathing*.—In nervous persons, and particularly in hysterical women, the inspiratory vesicular murmur is very apt to be broken into three or four distinct parts. This jerky breathing, which is heard over the whole lungs, disappears when the patient is told to take a deep inspiration, and is of no practical importance. But there is another variety of jerky breathing which differs from that just referred to in two particulars, viz.—(1.) it does not disappear with deep inspiration; and (2.) it is distinctly localised. This broken respiration is met with in cases of incipient phthisis, and is a sign of considerable importance, depending for its production upon some local obstruction to the entrance of air into the alveoli.

(3.) *Vesicular Murmur with Prolonged Expiration*.—It has been said that in healthy persons the expiratory murmur is frequently inaudible. When it is audible its duration is usually about one-third that of inspiration. When expiration exceeds this length we may conclude that either the lung tissue has lost its elasticity, or that there is some obstruction to the escape of air. One or other of these two conditions is met with in almost every affection of the lungs, so that in pulmonary disease a prolongation of the expiratory murmur is almost universal.

(4.) *Systolic Vesicular Murmur*.—Often during inspiration the breathing may be heard to be momentarily strengthened during the cardiac systole. When the heart contracts, the neighbouring portions of lung expand more rapidly to take the

place formerly occupied by the heart in diastole, and thus the systolic strengthening of the inspiration occurs. Its presence has not as yet been shown to be of any diagnostic significance.

**Bronchial Respiratory Murmur.**—The second great variety of respiratory murmur is that which is known as laryngeal, tubular, or bronchial. It can be heard in perfection when the stethoscope is placed over the larynx or trachea. Its peculiar character may be imitated by arranging the position of the mouth and tongue to utter the guttural “Ch,” and then breathing quietly out and in. The expiratory portion of the murmur is as long or longer than the inspiratory, and usually somewhat lower in pitch.

Bronchial breathing cannot be heard, in health, over the chest generally. Its area is confined to the larynx and trachea in the neck, and to the interscapular region close to the vertebral column\* (from the seventh cervical to the third or fourth dorsal vertebra), opposite to the bifurcation of the trachea, where, however, its special character is not so well marked as in the former situations.

*Mode of production of the Bronchial Respiratory Murmur.*—The air passing in and out of the chest with the movements of respiration, encounters at the glottis a considerable narrowing of the tube through which it is flowing, and in consequence vibrations arise in the immediate neighbourhood of the narrow point, which are of sufficient rapidity to be audible as a murmur. Underlying this vibrating point, however, there is the air column contained in the trachea and bronchi, which is set in vibration by sympathetic resonance, and thus the glottis murmur is augmented and reinforced. It is in this manner, in all probability, that the bronchial murmur in healthy persons is produced. It can readily be understood how, when the

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\* It is usually more distinct on the right side than on the left, owing to the greater calibre and more superficial position on the right bronchus.

opening of the glottis is narrowed by such a pathological process as croup, the murmur is louder and higher in pitch.

To the vibrations at the glottis, and those of sympathetic resonance in the broncho-tracheal air column, other vibrations may, however, be added in consequence of pathological changes in the air passages. When the lumen of the trachea is narrowed, as the result of the pressure of a tumour, vibrations and a consequent murmur arise at the stenosed point, and are reinforced by the underlying air-column just as the glottis murmur is; and even when the mucous membrane of the trachea and bronchi becomes swollen and roughened by catarrhal processes, the character of the bronchial murmur changes, and it becomes harsh—the result, no doubt, of local vibrations.

#### *The Bronchial Murmur in Disease.*

As I have already said, the bronchial murmur is only audible in health over the larynx, trachea, and less distinctly between the shoulder-blades. It cannot be heard over the chest generally, partly because it is overpowered by the vesicular murmur, and partly because inflated lung tissue is a very bad conductor of sound. This murmur becomes audible, however, under two varieties of pathological conditions as follows:—

(1.) *When the lung tissue becomes condensed*—provided that the condensation is extensive, and lies at or close to the surface of the lung, and contains besides a large and unobstructed bronchus—the vesicular murmur disappears over the condensation, and the bronchial murmur is conducted to the surface and becomes audible. These conditions are fulfilled in the case of acute pneumonia (stage of hepatisation), and in all the varieties of chronic phthisis. Bronchial breathing is therefore heard over hepatised lung, and wherever phthisical consolidation is of sufficient extent. It also occurs when the lung tissue is consolidated as the result of compression and collapse—as, for example, above the level of a pleuritic effusion; but it is not by any means always met with under such conditions,



for the pressure of the effusion must be sufficient to cause collapse of the air-cells, and yet not sufficient to obliterate the bronchi.

(2.) *In pulmonary cavities.* Bronchial breathing may be heard over vomicae, provided that they are superficial, have smooth walls, are surrounded by condensed tissue, and freely communicate by means of a bronchus with the air in the trachea. In certain cases it may be possible to judge roughly of the size of the cavity by the pitch of the bronchial murmur heard over it, since the air rushing into the cavity excites sympathetic resonance in it—that is, calls forth its special tone, which corresponds to the size of the resonating cavity, and this, if loudly enough heard, gives a guide to its capacity.

It is not, I think, desirable to subdivide bronchial breathing into a number of different varieties, as such a course only tends to cause confusion, without apparently promoting any useful purpose. It may, however, be well to mention that many writers recognise a modification of bronchial respiration, which Laennec named “cavernous breathing,” in which the air appears to the ear of the auscultator to pass into a large hollow space. It is not, nevertheless, characteristic of the presence of a vomica.

There is, however, one special variety of bronchial breathing to which attention must be directed, viz.:—

#### *Amphoric Respiration.*

The peculiar character of this variety of bronchial breathing is perfectly reproduced by blowing into an empty jar or bottle, and its mode of origin is similar to that of the sound so obtained. Amphoric breathing occurs under two pathological conditions—(1) pulmonary excavation, and (2) pneumothorax, as follows:—

(1.) *Pulmonary Excavation.*—The cavity must be of very considerable size, with smooth firm walls, and must lie super-

ficially. It must contain air, and must be in free communication with a bronchus. In such vomicæ the sonorous waves excited by the respiratory current\* are reflected again and again from the smooth walls, and so come to have an amphoric character, the prime tone being comparatively low in pitch, and the upper partials high and ringing.

(2.) *Pneumothorax*.—When air escapes into the pleural sac and distends it, the lung tissue becomes compressed; and if this pressure be sufficient not only to drive the air out of the air-cells, but also to cause collapse of the bronchi, no amphoric breathing occurs. But if the fistula by which the air has entered becomes closed before the pressure has become sufficient to obstruct the bronchi, the bronchial respiration will be conducted to the immediate neighbourhood of the large air cavity in the pleura, in which, by sympathetic resonance, sonorous vibrations will be excited. These vibrations, owing to the physical conditions met with in pneumothorax (smooth, firm walls, &c.), will have an amphoric character. If some quantity of serum or pus be present, along with air, in the pleural cavity, the pitch of the amphoric sound will vary according to the position of the patient, for the reason already mentioned.

Thus far I have described the two great classes of respiratory murmur, the bronchial and the vesicular. Between these two, however, there lies an intermediate variety, which may be called

*Broncho-vesicular Breathing.*

There may occasionally be heard a respiratory murmur which even the most practised ear cannot define as being either bronchial or vesicular. This murmur may be produced in healthy persons when they are directed to breathe superficially.

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\* As has been formerly explained, the sonorous waves formed in a pulmonary cavity, by the respiratory air-current, are the result of sympathetic resonance.

In disease this variety of breathing is usually the result of partial blocking up of the bronchi leading to the part of the lung examined, or to the interposition of some badly conducting substance between the lung and the stethoscope, such as pleural effusion, tumour, or even œdema of the chest-wall.

This broncho-vesicular murmur is only of diagnostic value when localised in one particular part of the chest, particularly when it is confined to one apex. In the latter case it points to the probability of commencing phthisical change.

## CHAPTER XXII.

### Respiratory System—(*continued*).

#### ADVENTITIOUS SOUNDS ACCOMPANYING RESPIRATION.

IN health the respiratory murmur is not accompanied by any other sound, but in the great majority of diseases of the lungs, at some part of their course, there become audible certain abnormal or adventitious sounds which are collectively known under the term *râles*. Inasmuch as certain of these *râles* give to the ear the impression of being caused by the bursting of air bubbles in a fluid, while others have a dry snoring or whistling character, they have been divided into two classes—moist and dry *râles*. Although this division is not scientifically accurate, some of the apparently moist sounds beings in reality formed without the presence of fluid, and certain of the dry *râles* owing their production to the presence of a more or less viscid secretion, yet the division is clinically useful, and ought not, I think, to be discarded.

Physicians differ much in regard to the nomenclature of these *râles*, and as a rule they have been too minutely subdivided. For all practical purposes the following classification will be sufficient :—

1. Moist *râles*—
  - (a.) Crepitation.
  - (b.) Fine bubbling *râles*.
  - (c.) Coarse bubbling *râles*.

2. Dry râles\*—
  - (a.) Sonorous.
  - (b.) Sibilant.
3. Pleuritic friction.

### Moist Râles.

#### *Crepitant Râle.*

The peculiar fine moist râle, which Laennec described under this name, has been compared to the sound produced by rubbing a lock of hair between the fingers close to the ear, or to the crepitation of salt when thrown upon the fire; but, as Eichhorst points out, both these sounds are too coarse, and crepitation may be more closely intimated by firmly pressing the moistened thumb against the forefinger, and then suddenly separating the two surfaces, close to the ear.

Although crepitation is probably sometimes due to the bursting of fine bubbles in the very smallest bronchioles, it commonly arises from the sudden separation of the alveolar walls, which have become adherent either to each other or to a mass of viscid secretion in the air cell. It is typically met with in the first stage of pneumonia, of which it is a most important sign, and it also occurs in pulmonary collapse and cedema.†

Crepitation occurs almost invariably only during inspiration, and is usually limited to the latter part of it alone. The individual crepitations of which it is composed are characteristically uniform in size, and are unaffected by the act of coughing.

Occasionally in health a momentary crepitation may be heard, usually at the lower posterior border of the lung, but sometimes also at the apex, when a deep inspiration is made, more especially when the patient has been lying on his back,

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\* Sometimes the term *rhonchus* is reserved for the dry sounds.

† In cedema bubbling râles are superadded, owing to the bronchi being filled with fluid, and hence the uniformity of the crepitation is lost. The sound is thus easily distinguished from the crepitant râle of pneumonia.

and the respiration has been very quiet for some hours. A knowledge of this fact will prevent any mistake.

*Fine Bubbling Râles.*

*Coarse Bubbling Râles.*

These two varieties of râles being closely associated, they may conveniently be considered together. The difference in the size of the bubbles in each case depends somewhat upon the quantity and quality of the fluid in which they originate, but chiefly upon the size of the space. The finer bubbling râles arise for the most part in the smaller bronchi, the coarser in the large bronchi, in the trachea, or in pulmonary cavities. In the great majority of cases in which these bubbling râles are heard, they vary in size, and are therefore spoken of as irregular, in contradistinction to the regular fine crepitant râle which has just been described.

Arising in fluid, as these bubbling râles do, we would naturally expect that they would be found most abundantly in the lower portions of the lung—the fluid obeying the law of gravity—and this is generally the case, the base of the lung posteriorly being their most common seat. When, on the contrary, they are heard most abundantly at the apices, and still more when they are exclusively met with there and persist for some time, the condition is one which must be looked upon with considerable gravity, pointing as it does to a local cause, which in the majority of cases is some form of pulmonary phthisis.

The finer bubbling râles in the smaller bronchi occur chiefly at the height of inspiration and the beginning of expiration, while coarse bubbling may be heard both during expiration and inspiration, being then continuous. In both cases a severe fit of coughing may remove the râles for the time. Their amount and intensity depend upon the quantity of fluid, the nearness of the bubbling to the surface, and the strength of the respiration.

In so far as the properties of these bubbling râles, which have as yet been described, go, their presence only informs us

that the air current encounters fluid in the respiratory passages, through which it bubbles. We now come to certain qualities in the tone of these râles, which give an indication of the condition of the surrounding pulmonary tissue. If the lung tissue around the point at which the bubbling is taking place is consolidated, the râles assume a clear musical high-pitched quality, and are termed *resonant*. Whenever such râles are heard we may conclude, with safety, that consolidation is present (although their absence does not permit of the exclusion of such a condition), and in fact resonant bubbling has a significance exactly similar to that of bronchial breathing. When the râles occur in a large cavity with smooth walls, and near to the surface of the lung, they assume a peculiarly clear metallic character—the *metallic tinkling*\* of Laennec. These râles are very musical, and have a high pitch which can readily be determined; and in regard to their physical cause and the conditions under which they occur, they stand in close relation to amphoric breathing and resonance. Similar resonant râles may be heard over large air cavities which lie in close proximity to the lungs, such as a pneumothorax, or even the stomach or intestine when distended with air. In such cases it is not necessary that the râles arise in pulmonary cavities; they may originate simply in the bronchi, and the neighbouring air cavity may act as a resonator, reproducing and intensifying the sound.

**Dry Râles** are produced in the air passages by any pathological process which narrows their lumen, the most common being the accumulation of viscid secretion and the swelling of the mucous membrane. When they arise in the larger bronchi they are low-pitched and snoring (sonorous râles), when in the smaller tubes they have a whistling character (sibilant râles). Both varieties occur chiefly during inspiration, the snoring râles

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\* This tinkling was supposed to be sometimes produced by the dropping of fluid from the roof of a cavity, but this manner of production would seem to be exceedingly doubtful.

at its commencement, the sibilant not till towards its termination.

Both these varieties of dry râle occur in cases of bronchial catarrh, whether acute or chronic, primary or secondary, and according as they are sonorous or sibilant we may infer that the larger or the smaller bronchial tubes are affected.

The presence of pulmonary consolidation round the point at which these râles occur imparts to them a ringing musical character, but as their quality is in any case musical, this change has not anything like the diagnostic value which it possesses in the case of moist râles.

**Pleuritic Friction.**—The gliding of one pleural surface over the other, which occurs normally with each respiration, is accomplished without any sound; but when, as the result of pleurisy, the surfaces become rough and uneven, sound of friction becomes audible. This sound varies from the lightest rubbing, only perceptible with difficulty, to loud creaking, which can readily be made out on palpation, and which the patient himself both feels and hears. The sound is usually broken up into portions of greater and less intensity, and while it is sometimes audible throughout the whole of both respiratory phases, it is usually limited to the latter portion of inspiration. In cases of pleurisy the friction sound becomes audible whenever the process has advanced sufficiently far to cause considerable roughness of the pleural surfaces, and it of course disappears when those surfaces are separated by effusion, to reappear when absorption of the fluid has taken place. Although the friction sound may sometimes be audible over a great part of the lung, it is usually limited to a small area, and occurs most frequently in the axillary region. When the friction sound is heard at the apex of the lung it points with great probability to phthisis.

With regard to differential diagnosis, the pleuritic friction-sound is sometimes closely simulated by râles in the air passages.



Attention to the following points will usually suffice to distinguish them :—

<i>Râles.</i>	<i>Friction.</i>
Modified by coughing.	Not modified by coughing.
Not affected by pressure of the stethoscope.	Intensified by pressure of the stethoscope.
Usually heard over wide area.	Usually localised.

From pericardial friction the sound of pleuritic friction may be distinguished by causing the patient to hold his breath, when the latter will disappear and the former continue.

**Auscultation of the Voice.**—In a former chapter the *fremitus*, or vibration of the chest walls, produced by the act of speaking, has been described. As regards its causation and the various pathological conditions under which it is enfeebled or intensified, the resonance of the voice closely corresponds to the vocal *fremitus*.

When the stethoscope is applied to the chest while the patient speaks, only a soft indistinct murmur is to be heard, provided that the lung is healthy. Over the larynx and trachea, however, this vocal resonance is much intensified, and it is almost as though the words were spoken into the opening of the stethoscope. The intensification is termed *bronchophony*.\*

Before speaking of changes in the vocal resonance produced by pathological conditions connected with the lung, it may be as well to repeat what was said in connection with vocal *fremitus*—viz., that the vibrations of the voice over the thoracic parietes, audible as well as perceptible to palpation, depend

\* The highest development of bronchophony was termed *pectoriloquy* by Laennec, but there is no fundamental difference between the two, and there seems to be no good reason for adding an additional term to the auscultatory nomenclature, which is already sufficiently complicated.

for their intensity upon the loudness and depth of pitch of the voice, and upon the thickness of the chest wall ; that the vocal resonance (like the corresponding fremitus) is more distinct in men than in women ; and that it is almost invariably louder on the right side than on the left, owing to the larger calibre of the right bronchus.

Bearing these points in mind, we may now consider the changes in the vocal resonance which result from pulmonary disease.

*Enfeeblement of the Vocal Resonance.*—The vocal resonance is diminished when the lung is separated from the chest wall by collections of liquid\* or air in the pleural cavity, and when the bronchi leading to the part of the lung in question have become blocked up with secretion.

*Intensification of the Vocal Resonance (Bronchophony).*—As has been already said, bronchophony occurs normally over the larynx and trachea down to the bifurcation of the latter in the interscapular region. When bronchophony occurs at other points of the chest it is pathological, and it then owes its origin to consolidation of lung tissue, and the consequent better conduction of the vocal vibrations to the chest wall. Bronchophony thus arises, along with bronchial respiration, in all diseases which lead to condensation—for example, in acute pneumonia, and in all the forms of phthisis. It is particularly noticeable over pulmonary vomicae, the resonance of the air in the cavity adding to the intensity of the vocal resonance, and imparting to it in addition a peculiar metallic character. As a whole, it may be taken that bronchophony has an exactly similar significance to bronchial respiration.

It has been said that pleural effusions diminish or even suppress the vocal resonance ; but this is not always the case. Baccelli pointed out, in 1875, that the resonance of the whispered voice was often heard very clearly over pleural effusion. This *pectoriloquie aphonique* he held to occur only

\* With the exceptions to be presently mentioned.

when the fluid was homogeneous (serous effusion), and not when the effusion was heterogeneous (pus). There can be no doubt that this sign is very frequently present in such cases, but recent observations\* have failed to confirm its value in so far as the discrimination between serous and purulent effusions is concerned.

Under certain conditions, bronchophonic vocal resonance assumes a very peculiar nasal quality, resembling the noise produced by speaking against a comb covered with paper, and which, from its supposed resemblance to the bleating of a goat, Skoda termed

*Aegophony*.—This variety of bronchophony is most commonly met with in cases of pleuritic effusion, near the upper margin of the fluid, and usually close to the lower angle of the scapula. As to the exact manner of its causation there is some doubt, but most observers are agreed that it depends upon compression and partial obstruction of the bronchi. Its diagnostic value does not materially differ from that of ordinary bronchophony.

**Hippocratic Succussion.**—We must, in conclusion, refer briefly to this sign, which was described by Hippocrates, and which, although rarely met with, is of considerable interest.

If, in cases of pyo-pneumothorax, the ear be applied to the chest, and the patient shaken, a ringing splashing sound may be heard, which is the sound in question. The splashing noise becomes intensified by the resonating air cavity above the fluid in the way I have already described. This succussion sound may also be heard when there is a very large excavation in the lung tissue partially filled with fluid.

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\* Dr. Douglas Powell, Tr. International Med. Congress, 1881.

## CHAPTER XXIII.

### *Integumentary System.*

THE study of the affections of the skin is of great importance to the physician, not merely on account of the frequency of their occurrence, the distressing severity of their symptoms, and the deformities which they leave behind them in their course, but also because they are frequently symptomatic of the general condition of the system, mirroring forth with fidelity not a few grave systemic diseases, and still more often the many slighter disorders which it is important to recognise and to check at their outset, and of which the physician may have no other indication. Among the serious diseases of which the condition of the skin gives evidence, it is hardly necessary to mention syphilis, scrofula, as well as all the members of the important group of exanthemata. Then, again, it is well known that errors in diet, and disorders of the digestive functions generally, are apt to cause various forms of skin disease (urticaria, acne, &c.), and without going further into detail in illustration of this point, it may finally be mentioned that many uterine affections, and even pregnancy itself may be accompanied with blotches on the skin (chloasma uterinum), which in certain circumstances may be a symptom of not a little importance.

**Subjective Symptoms** are of comparatively little diagnostic importance in cases of skin disease, although to the patient they are often very distressing. Sensations of heat attend all the inflammatory processes in the skin. Hyperæsthesia and anæ-

thesia are met with in the various cutaneous neuroses, and shooting pain is a prominent symptom of herpes zoster, frequently preceding the eruption, and persisting for some time after its disappearance. But of all the subjective symptoms of skin disease, the most distressing is itching. It is very common as a result of the presence of parasites, but frequently occurs independently of such agents in cases of eczema and of pruritus.

**Objective Symptoms.**—A patient suffering from skin disease ought to be examined in a well-lighted and warm room, preferably by daylight, and if a male, the whole surface of the body ought to be viewed by the physician if the case is at all important.

The general condition of the skin as to colour and moisture, the deposit of subcutaneous fat, and the presence of cedema and of emphysema are points which have been already considered in Chapter I., and need not be again referred to.

There only remain, therefore, for our consideration, the various eruptions which occur on the skin.

### ERUPTIONS.

In considering skin eruptions, there are four main points to which attention should be directed, and under which the facts observed may be satisfactorily classified.

#### 1. *The Distribution and Configuration of the Eruption.*

When the whole surface of the body is covered, the eruption is said to be *universal*; when it is irregularly scattered over various parts of the body, it is said to be *diffused*. The configuration of the individual lesions is commonly defined by such terms as *punctate* when of the size of pin-heads, *guttate* when resembling drops of water, *nummular* when of the size of coin, &c.

The distribution of the lesion is often of considerable diagnostic importance. For example, psoriasis is usually only

found on the extensor aspect of the limbs, whereas the macular, papular, and squamous syphilides, when they appear on the limbs, are seen on the flexor surfaces. Then, again, certain lesions, as herpes zoster, follow the course of nerves. Lupus is usually found on the face, erythema nodosum on the leg, seborrhœa sicca on the scalp, acne on the face and back, and the scabies insect selects for its burrows by preference the skin between the fingers.

### 2. *The Elements of the Skin involved.*

Careful inspection of the skin will inform the physician as to the condition of the epidermis, the hair, the orifices of the hair follicles, the sebaceous and the sweat glands. Palpation of the skin will further give information regarding the condition of the true skin, whether it be infiltrated or not. Pressure with the finger on a pigmented spot will show whether the coloration is due to hæmorrhage or to hyperæmia, for in the latter case the colour will disappear on pressure. Further, if the skin be covered with crusts, the removal of these by means of the finger will display the condition of true skin, which in cases of eczema, for example, may be found to be moist, in seborrhœa dry, and in psoriasis bleeding. We have further in the diagnosis of skin cases to rely upon the evidence afforded by the microscopic examination of the hair, crusts, &c., as will be more particularly pointed out when we come to speak of the ætiology of such affections.

### 3. *The Type of Eruption.*

The very numerous forms assumed by skin eruptions may be defined and described as follows :—

(a.) *Macules (Maculæ).*—These consist in morbid changes in the colour of the skin, which are circumscribed, and do not involve the whole cutaneous surface, and which are neither elevated above nor depressed below the surface of the skin. Such macules may arise in very various ways. Sometimes, as

in erythema fugax, they are occasioned by hyperæmia, and then the colour disappears on pressure; sometimes by hæmorrhage, as in purpura; by increase or decrease of the normal pigment; by exudations into the tissues of the true skin, as in syphilides; and, finally, they sometimes arise from increase in the size and number of the blood-vessels, as in nævi.

(b.) *Papules* (*Papulæ*) are small firm elevations above the surface of the skin, varying in colour, and arising in very different ways. The simplest form of papule is seen in the *cutis anserina* or goose-skin, due to the contraction of the muscles of the skin. Pathologically, papules form as the result of hypertrophy of the papillæ (ichthyosis), of cell proliferation in these structures (lupus, syphilis), or of inflammation of and consequent exudation into these papillæ, as in eczema papulosum. Extravasation of blood into the skin may give rise to papules, as is seen in purpura papulosa. Papules may also be formed in connection with the sebaceous glands (miliun, comedo, acne), or by accumulation of epidermic cells round the hair follicles, as in lichen pilaris.

(c.) *Tubercles* (*tubercula*) are simply exaggerated papules. They are usually occasioned by cell proliferation, and occur as the result of syphilis, carcinoma, leprosy, &c.

(d.) *Tumours* (*phymata*) hardly require a definition here. They may be of considerable size, even as large as a child's head. Examples are seen in molluscum, and in the various cystic growths met with in connection with the skin.

(e.) *Wheals* (*pomphi*) are flat, irregularly-shaped, firm elevations on the skin, pale in the centre, red at the edges, and which are very fugitive. Wheals are typically seen as the result of the sting of the nettle, and in urticaria. They result from sudden effusion of the serum into the papillæ, and swelling of the cells of the rete malpighii, produced probably by vaso-motor changes.

(f.) *Vesicles* (*vesiculæ*) are small rounded elevations of the cuticle, varying in size up to that of a split pea, containing serous, sero-purulent, or bloody fluid, and either lying between

the mucous and horny layers of the epidermis, or in connection with the hair follicles, or with the sebaceous or sweat glands.

(g.) *Blebs (bullæ)* only differ from vesicles in point of size, being larger than a split pea.

(h.) *Pustules (pustulæ)* are elevations of the epidermis, similar in shape to vesicles and blebs, but containing pus. They are sometimes found in substance of the true skin (boils), in connection with hair follicles (as in sycosis), or in sebaceous glands (acne), or between the mucous and horny layers of the epidermis, as in small-pox. Pustules usually dry up (with or without bursting) into yellow or brownish crusts, and very often leave permanent cicatrices, if the tissues of the true skin have been involved.

Thus far we have been considering what are called the *primary* lesions, and we now pass to those which are secondary.

(i.) *Excoriations* are breaches of the continuity of the skin, often produced by the patient's nails. They give an indication of the amount of itching which is present. When lice are present (phthiriasis), the marks of the scratches are long and straight; in pruritus, they are short and irregular; and in scabies, small and round.

(j.) *Scales (squamæ)* are portions of epidermis which have become separated by diseased processes in the skin. The deeper and more severe the inflammation, the more marked is the desquamation. The scales may be thrown off as fine bran-like particles (as in prurigo, pityriasis, measles, &c.), or as thin flakes or thick plates (in psoriasis and eczema); or the epidermal layer to be thrown off may, as in scarlatina, separate as a whole, forming a more or less perfect cast of the fingers, or even of the whole hand.

(k.) *Crusts* are formed by the drying up of the products of skin disease, serum, pus, blood, &c. When chiefly composed of pus, they have a greenish colour; when mixed with blood, the crusts are brown or black. The firmest and hardest crusts are those met with in syphilitic processes (rupia), when they



often assume a form closely resembling that of a limpet shell. The crusts of favus are yellow and cup-shaped.

(l.) *Fissures (rhagades)* in the skin may involve the epidermis alone, or both the epidermis and the true skin; or they may be seated in mucous membrane. They are usually found where the skin is normally furrowed—as, for example, on the palms of the hands and soles of the feet; at the angles of the mouth; where the upper lip and nose join; at the elbows and knees; at the anus, and in other similar situations. Fissures are found in cases of chronic eczema and inveterate psoriasis, in syphilis, and in scleroderma.

(m.) *Ulcers* are chiefly within the domain of surgery. Their size, depth, shape, situation, and general condition should be noted.

(n.) *Cicatrices* follow all diseases or injuries of the skin which involve loss of substance. The character of the scar is not indicative of the preceding disease; but sometimes the number or seat of the cicatrices may afford some indication of their cause.

#### 4. *The Etiology of the Eruption.*

Skin eruptions are much influenced by the age and sex of the patient, by the season of the year, and by climate. On these points, and on the heredity of many such diseases, we need not now dwell. Very frequently skin affections are the result of constitutional diseases—as, for example, purpura, scrofula, rickets, all the acute exanthemata, diabetes, &c. We have further to note that diseases of particular organs often give rise to skin eruptions. Disorder of digestion from improper diet (shell-fish, for example), or other cause, is frequently followed by urticaria and acne. In valvular disease of the heart, œdema and small hæmorrhagic extravasations (petechiæ) frequently occur. Bright's disease is often accompanied by pruritus, and sometimes by eczema. Many other instances, too numerous to mention here, will occur to the reader in which diseases of the different internal organs are

accompanied by skin eruptions which are more or less characteristic.

A class of eruptions with which it is very important that the physician should be familiar are those which result from internal and external use of certain medicines.

All counter-irritants—such as croton-oil, mustard, cantharides, tartar-emetic, iodine, turpentine, arnica, &c.—give rise to various forms of dermatitis; as do also the various aniline colours with which stockings are sometimes dyed.

The internal administration of medicines is occasionally followed by skin eruptions, a result which is most frequently due to some peculiar idiosyncrasy of the patient. Among these may be mentioned the acne pustules which follow the use of the bromides, the erythema (or even eczema) of the iodides, and the scarlatina-like efflorescence of chloral. Very characteristic of atropia-poisoning are the bright erythematous patches which appear on the chest and neck. Morphia sometimes gives rise to an erythematous eruption resembling that of scarlatina, and the administration of quinine is occasionally followed by a rash of the same description. The eruption of copaiba usually shows itself upon the extremities as a bright papular efflorescence, which is generally very itchy.

Among the causes acting locally in the production of skin eruptions may be mentioned (in addition to the external applications just noticed) the following:—Continued exposure to the heat of a strong fire is apt to give rise—in furnacemen and cooks, for example—to eczema. Those who work in acids or alkalis, and especially in aniline dyes, suffer much from eczema. Even the long soaking of the hands and arms in hot water and soap produces in washer-women a hardened, fissured, and even eczematous condition of the skin of these parts. The most important local cause of skin eruptions is, however, undoubtedly to be found in the irritation set up by the various parasites which infest the skin and hair. The diagnosis in such cases is closely bound up with the etiology, and they must be considered together. The parasites which affect the skin be-

long to both the animal and vegetable kingdoms. The most important of these are the following:—

*Vegetable Parasites.*

1. *Achorion Schönleinii*.—This parasite gives rise to the disease known as tinea favosa, or favus. While it occasionally attacks the nails, it is more usually found upon the scalp, where it gives rise to the formation of light yellow, dry, cupped crusts.



FIG. 29 — *Achorion Schönleinii* (after Dühring)

The hair follicle and hair are first attacked, and then the parasite spreads itself upon the surface of the skin. When a part of one of these crusts is examined with the microscope, after having been soaked in water and treated with acetic acid or an alkali, the parasite (fig. 29) is readily recognised. It consists (1) of spores; (2) of slightly elongated elements, which

are usually united in rows ; and (3) of mycelium, which is made up of long, branching, transparent filaments, which may or may not contain spores in their interior. In the favus crust there are always to be found, in addition, numerous micrococci and bacteria.

2. *Trichophyton*.—This parasite produces three forms of skin disease—*tinea circinata*, or ringworm of the body ; *tinea tonsurans*, or ringworm of the scalp ; and probably *tinea sycosis*, or *sycosis parasitica*, a similar affection of the beard and other hairy portions of the face. On the body, the trichophyton gives rise to considerable irritation of the skin, which results in the formation of circular circumscribed patches of various size, slightly elevated above the level of the skin, of a dull red colour, and usually covered with small branny scales, while round the edges there may be found vesicles, and sometimes even pustules.

On the scalp, ringworm shows itself as one or more circumscribed patches of a greyish or slightly ruddy colour. The hair of the affected parts is short, lustreless, easily drawn out, breaks readily, and the extremities are ragged and uneven. The skin is covered with numerous thin white scales, and occasionally with crusts.

On the beard and upper lip, the parasitic form of *sycosis*—which is probably caused by the trichophyton—at first exhibits characters closely resembling those of ringworm of the scalp. As the disease advances, however, the skin and deeper parts become inflamed and indurated, and, as a consequence, the affected portions become covered with characteristic tubercular elevations, and pustules occupy the hair follicles.

In all these situations the trichophyton presents similar microscopic appearances. In the case of *tinea circinata*, a few of the scales should be scraped off the patch with a penknife, laid on a microscope slide and examined, after the addition of a dilute solution of carbonate of potassium. In the other forms, a diseased hair should be extracted and examined, after the addition of liquor potassæ or chloroform. Whether in the hair

(as in fig. 30) or spread over the surface of the skin, the parasite will be found to consist of long, slender, jointed filaments (mycelium), together with small, round, highly refractive spores. The latter are most abundant in ringworm of the scalp, infiltrating densely the hair bulb, while the mycelium spreads up the shaft of the hair.\*

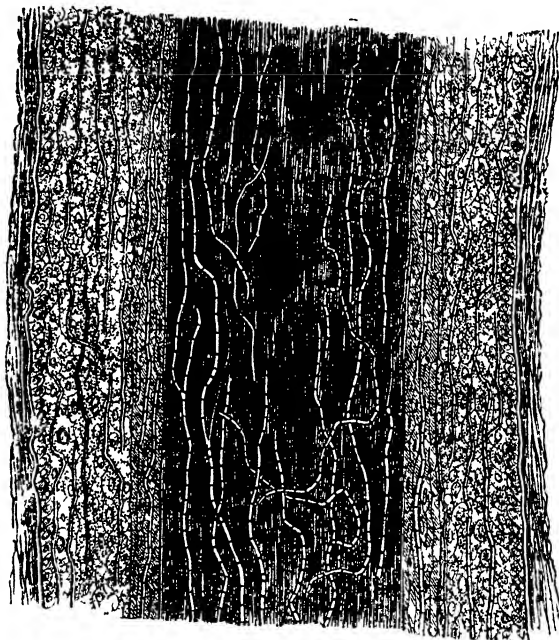


FIG. 30.—Hair affected with *Tinea Tonsurans* (after Neumann)

3. *Microsporon Furfur* is the parasite which gives rise to pityriasis versicolor. This disease is characterised by the presence on the skin (usually of the back and chest) of variously-

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\* It is very probable that a form of eczema which affects the inner surfaces of the thighs (eczema marginatum) also owes its origin to the presence of the trichophyton.

sized pale yellowish-brown or reddish patches covered with fine powdery scales. It is most frequently met with in those suffering from wasting diseases. The microsporon furfur consists of spores and mycelium. The spores are small, round or oval, highly refractive bodies, which tend to arrange themselves in groups in a manner which is very characteristic of this parasite. The mycelium consists of fine curved filaments which are usually short and are jointed together, forming a close network. In their interior spores are generally to be seen.

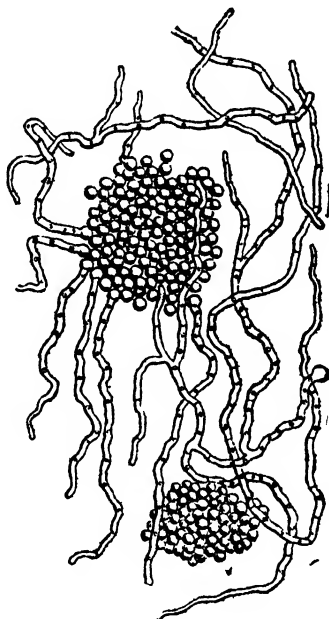


FIG. 31.—*Microsporon Furfur* (after Neumann)

This parasite is very readily detected by means of the microscope. A few of the scales on the surface of the patch should be scraped off with the penknife, placed upon a cover-glass, and treated with liq. potassæ.\*

#### *Animal Parasites.*

1. *Sarcoptes Scabiei*.—The skin disease—scabies, or the itch—which is caused by presence of this insect, consists of papules, vesicles, pustules, excoriations, fissures, crusts; in short, an eczema, in the neighbourhood of the burrows in which the

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\* The patches of baldness of alopecia areata are by some supposed to be parasitic, and to depend on the presence of the microsporon audouini, but there is considerable uncertainty on this point.

insects lie. Scabies is usually found between the fingers, but may spread over the body generally, the insect selecting, however, localities where the skin is soft and thin. It is the commonest of all skin diseases, and is very contagious.

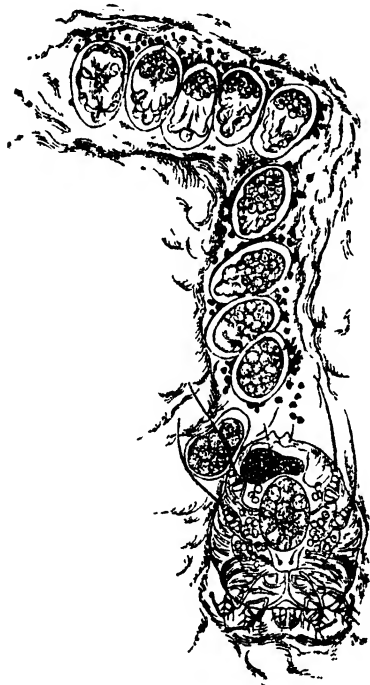


FIG. 32.—*Sarcoptes Scabiei* (after Neumann).

The female insect, which can be seen in fig. 32 lying at the end of its burrow, has an oval body marked with fine undulating lines, a small oval head, and possesses eight legs (the four front legs being furnished with suckers) and numerous bristles. The male insect does not give rise to the eruption, and is but seldom met with. The female, after being impregnated, bores through the horny layer of the cuticle perpendicularly, and then forms a burrow which runs horizontally in the mucous layer of the epidermis. This burrow, or cuniculus, can

often be seen by the naked eye as a whitish elevated line terminating in a minute speck, which is the insect. The cuniculus becomes filled with eggs, of which the insect lays about ten to fifteen, and with small black specks of excrement. The eggs are hatched in about ten days.

With a little trouble the itch insect can usually be secured, and the diagnosis thereby rendered certain. A needle should

be introduced into the burrow, which the insect will generally seize hold of, and on the point of which it may be removed.

2. *Pediculus*.—Three varieties of pediculi are met with on the human body, giving rise to the disease termed phthiriasis—the pediculus capitis, pediculus corporis, and pediculus pubis.

The head louse is an elongated ovalish insect of a greyish hue. From its head spring two antennæ, each consisting of five parts, and to the thorax are articulated six legs armed with strong claws. The oval whitish eggs are firmly fastened to the hairs of the patient's head by means of a viscid glue-like material. The irritation and itching of the scalp and the consequent scratching give rise to a severe eczematous condition, in which the serous, sanguineous, and purulent exudations matt together the hairs, and almost invariably cause enlargement of the neighbouring lymphatic glands.

The pediculus corporis, or pediculus vestimenti, as the insect is sometimes termed, resembles closely the head louse in structure, but is somewhat larger. The cutaneous lesions consist chiefly in long scratch marks, crusts, and papules, along with the minute red points where the insect has bitten.

The pediculus pubis, or crab-louse is smaller and more rounded than either of the other species. It infests chiefly the pubic region, and usually gives rise to considerable irritation.

3. *Pulex Irritans*, the common flea, need hardly be mentioned here, were it not that flea bites are occasionally mistaken for purpuric spots. Round the bite, however, there will be seen to be a hyperæmic areola, which is not met with in purpura.

4. *Demodex Folliculorum* is a harmless, worm-like parasite, which inhabits the sebaceous follicles of the skin of the face. If the contents of a prominent follicle be squeezed out and examined with the microscope, the demodex will frequently be found.



## CHAPTER XXIV.

### Urinary System.

#### SUBJECTIVE SYMPTOMS.

BEFORE proceeding to the consideration of the various changes met with in the urine in disease, which must always rank as the most important sign of urinary disorders, it may be well to note certain subjective symptoms which occur in such cases, and which often give very valuable indications.

**Pain** may be felt at different portions of the urinary tract, as follows :—

1. *At the end of the Penis.*—In calculus of the bladder pain is felt after micturition, because the rough stone then comes in contact with the bladder wall; it is referred chiefly to the extremity of the penis, and is increased by any sudden movement. In prostatitis, also, pain occurs after passing water, the bladder then contracting on the tender prostate. In women there is often severe pain felt during micturition at the orifice of the urethra, owing to the presence there of a small vascular growth.

2. *In the course of the Urethra.*—When the urethral canal is narrowed by stricture, pain is felt at the constricted point during micturition. In urethritis, also, the pain during the passing of water is referred to the urethra. When the urine is highly acid, concentrated, or contains gravel, urethral pain also occurs during micturition.

3. *Over the Bladder in the Supra-Pubic Region.*—This is the common seat of the pain of cystitis, which, it is to be observed, occurs before micturition, and is relieved by that act. In acute cases pain may also be felt deep in the perineum.

4. *In the Loins.*—In cases of pyelitis and of renal calculus there is usually dull aching pain over the loins, which is increased on pressure, and which in the latter disease occasionally passes into violent paroxysms, the pain shooting down the ureters to the testicle and inside of the thigh.

**Frequency of Micturition.**—Wherever the urine is large in quantity, as in diabetes and the waxy form of Bright's disease, for example, there is frequency in micturition. This symptom, however, also occurs in very many other urinary disorders. In all inflammatory conditions of the prostate and bladder, in pyelitis and nephritis, in calculus of the bladder or kidneys, the urine is frequently voided. It is particularly to be noticed that in the cirrhotic or contracting form of Bright's disease, and in hypertrophy of the prostate, the calls to micturate are frequent, and occur chiefly during the night.

### *The Examination of the Urine.*

In such a work as this it is of course quite impossible to give anything like an exhaustive account of the many changes which take place in the urine in health and disease, or of the various methods of analysis which have been applied to that secretion. All that I shall attempt to do will be to enumerate the more ordinary and clinically significant changes which occur, and the simpler methods of analysis, such as may be carried out by the physician, excluding those which require the more complicated apparatus of a chemical laboratory.

In the present chapter we will consider the general condition of the urine as to (1) quantity, (2) colour and transparency, (3) odour, (4) specific gravity, and (5) reaction.

**Quantity of the Urine.**—While varying according to the quantity of fluid drunk, the amount of the pulmonary and cutaneous transpiration, and of the alvine discharge, the average quantity of urine voided in twenty-four hours may be taken to be in the adult from 35 to 60 ounces.

The quantity is diminished in all febrile diseases, in heart affections when compensation is lost, in cases of collapse, and generally in all those conditions in which much fluid is passing out of the blood, such, for example, as profuse diarrhoea or perspiration, the rapid accumulation of serum in the pleuræ or peritoneum, &c. Further, there is scanty urine in the inflammatory form of Bright's disease whether there be inflammation of the tubules or of the glomeruli. The urinary flow may be completely suppressed in cases where the ureters are occluded by the impaction of calculi, or by the pressure of morbid growths.

The urinary flow is *increased* by the administration of diuretics. It is greatly augmented in cases of diabetes insipidus and mellitus, chiefly owing to the large quantities of water which such patients drink. In the cirrhotic, or contracting form of Bright's disease, the quantity of urine secreted is increased in the later stages, when the heart has become hypertrophied, and the vascular tension increased. On the other hand, in the waxy form (as was first pointed out by Professor Grainger Stewart) polyuria is an early symptom, often occurring even before the presence of albumen can be detected.

**Colour of the Urine.**—The urine owes its colour to the quantity of pigment it contains, and to the amount of its concentration—very dilute being pale, very concentrated having a dark brownish-red colour. For convenience of comparison, Vogel's standard scale of colours is usually adopted. The various tints are grouped as follows:—

Yellow Urines,	{ Pale Yellow, Bright Yellow, Yellow.
Red Urines,	{ Reddish yellow, Yellowish-red, Red.
Dark Urines,	{ Brownish-red, Reddish-brown, Brownish-black.

In order to obtain uniform results as to colour, the urine should be examined in a glass, the diameter of which is about four inches ; and if not absolutely clear, the urine must be filtered before its colour is noted.

Very pale urines are met with in healthy persons after copious draughts of water, and, further, in cases of diabetes and of anæmia, and after hysterical paroxysms. Highly-coloured urines occur in the febrile state, and under other pathological conditions, which will be mentioned more particularly hereafter.

What the pigments of normal urine are, is still a matter of doubt. There are, however, two pigments, both of which must be looked upon as pathological, uro-bilin and uro-erythrin, about which a few words must be said.

*Uro-bilin* is a reddish pigment, first described by Jaffé\* which is found in considerable quantity in the urine of fever, and sometimes in that of jaundice. In normal urine, when first voided, it does not occur, but it often appears after it has been allowed to stand for some time in contact with the air. The chief interest of uro-bilin is derived from its relations with, on the one hand, bilirubin, which as Maly† has shown, when treated with sodium-amalgam, yields a body which he named hydro-bilirubin, and which is identical in all its characters with uro-bilin ; and on the other hand, with blood pigment, for Hoppe-Seyler has pointed

\* Virchow's *Archiv.*, vols. xlvii. and xlviii.

† *Centralbl. f. d. Med. Wissensch.* 1871.

out that when hæmatin in alcoholic solution is treated with tin and hydrochloric acid, uro-bilin is formed. The detection of uro-bilin is usually easy, and depends chiefly upon the three following points:—(1.) Examined with the spectroscope, most urines which contain uro-bilin show an absorption line between Fraunhofer's lines *b* and *F*, which is not very well defined, and which shades away towards *F*. Sometimes, however, the spectrum cannot be made out in the urine itself. In such cases, if the urine be shaken up with ether, the ethereal solution of the pigment will show the spectrum clearly. (2.) When a small quantity of chloride of zinc is added to an alkaline solution of the pigment, a green fluorescence appears. (3.) The addition of ammonia to the urine itself, or to an acid solution of uro-bilin, changes the reddish colour into clear yellow.

*Uro-erythrin* is a pinkish-red pigment (the purpurin of Bird) which often appears in the urines of fever, and of cirrhosis of the liver, and which attaches itself to precipitates of urates and of uric acid, giving the sediment a brick-dust colour. This deposit, however, often occurs in otherwise healthy persons, from errors in diet and other slight causes.

*Melanin*, the black pigment which is found in the urine in cases of melanotic cancer, may at times possess some diagnostic significance.

The administration of certain drugs is followed by alteration in the colour of the urine. Thus, after the absorption of carbolic acid, the urine becomes of a dark greenish-brown colour, due to the presence of an oxidation product of hydrochinon. Rhubarb and senna (chrysophanic acid) colour the urine a deep brownish-yellow, which changes to bright red on the addition of an alkali. Logwood imparts a red tinge, and santonin a bright yellow which changes to orange when ammonia is added.

The presence of blood and bile pigments in the urine will be considered hereafter.

**Transparency.**—Normal urine, when freshly passed, is

almost invariably transparent; but when allowed to stand, clouds of mucus form in it, which, at the end of twelve hours, will be found to have sunk to the bottom of the vessel. In highly-concentrated urine, and especially in that of the various feverish processes, a dense cloud of urates forms after cooling has taken place, which, as well as the other urinary sediments, will be considered farther on in these pages.

**Odour.** — Freshly-passed normal urine has a faint odour peculiar to itself, which gradually disappears. When it becomes alkaline, an ammoniacal odour develops itself in the urine. When blood or pus becomes added, the urine has a peculiarly offensive odour from its rapid decomposition.

Turpentine, when inhaled or taken internally, imparts an odour of sweet violets to the urine. Copaiba, cubebs, tolu, and asparagus, also communicate a characteristic smell. Finally, in diabetes mellitus, the urine has a faint, sweetish odour, which, if acetonæmia develops itself, comes to resemble that of chloroform.

**Specific Gravity.**—The specific gravity is usually and most conveniently estimated by means of a urinometer. The instrument is dipped into the urine and allowed to float, the point at which the level of the surface of the urine cuts the graduated stem being read off, and thus the specific gravity is ascertained. One or two precautions must, however, be taken. The urinometer must be carefully dried before use, as drops of water adhering to the upper part of the stem tend unduly to depress it. It must also float completely clear of the edge of the vessel, and the surface of the urine must be free from air-bubbles, which, if present, can be readily removed by means of filter-paper. As urinometers are graduated for a temperature corresponding to that of an ordinary room, observations must not be made on urines until they have cooled down to that point.

The urinometer scale commences at 1000, the specific gravity

of distilled water, and usually goes up to 1050. The average specific gravity of normal urine may be taken to be from 1015 to 1025; but readings both above and below these limits are quite consistent with perfect health. The specific gravity of any urine expresses, of course, the quantity of solids which that urine contains in solution. Thus, if we find it in any particular instance to be, let us say, 1025, we know that there are present solids in such quantity as to suffice to raise the weight of a litre of distilled water from 1000 grammes to 1025.

From the specific gravity so obtained, it is possible roughly to calculate the quantity of solids present in the urine. This may be done by means of the very simple formula given by Trapp, which consists in multiplying the two right hand figures by 2, the result being the amount of the solids in 1000 parts of the urine.

From what has been said, it is clear that as the specific gravity of the urine depends upon the proportion of solids to fluid, it will be affected by changes in the quantity of either. Thus, after copious imbibition of water, the urine of healthy persons may have a specific gravity as low as 1002; and, on the contrary, after profuse perspiration, it may rise to 1040. We must thus take into account the *quantity* of the urine passed in twenty-four hours before we allow ourselves to judge what importance is to be attached to the specific gravity. When the quantity is large, we find, if the urine be normal, a low specific gravity; whereas, when the flow is scanty, the specific gravity is high. If, however, we meet with a urine which, while large in quantity, possesses a high specific gravity, or one which, while small in amount, is low in gravity, then the fact may in each case be noted as distinctly pathological.

Pathological urines may be classified as follows:—

*High Specific Gravity.* is found after copious perspiration, vomiting or purging, owing to the consequent concentration of the urine. At the commencement of all acute feverish

diseases, the specific gravity of the urine is high, running up even to 1035, and this, owing, in part, to diminished watery excretion, but also, in great measure, to the increased elimination of urea, sulphates, and phosphates which then takes place. Much more marked and important, however, is the increase of specific gravity met with in the urine of diabetes mellitus. In this disease we find large quantities of urine being passed, the specific gravity of which varies from 1030 to 1060, its height being due to the presence of grape sugar.

*Low Specific Gravity*, when not due to great dilution of the urine, is commonly the result, either of some disturbance of the secreting apparatus of the kidney (Bright's disease, circulatory disease, &c.), or of general interference with nutrition (anæmia, cachexia, &c.), in both cases arising directly from the defective elimination of the urinary salts, particularly urea and its compounds.

**Reaction.**—The reaction of the urine may be tested by means of blue and red litmus paper. Normal urine is acid when fresh, very rarely neutral or alkaline, the acidity being due to the presence of free acids—such as lactic, oxalic, hippuric, and acetic—and acid salts. After a meal, the urine loses in acidity, sometimes becoming neutral, or even alkaline; but it very rapidly regains its former character. The effect of both warm and cold baths is to render the urine alkaline; and the same result is produced much more powerfully by the action of such alkaline medicines as the bicarbonates and acetates of potassium and sodium. The effect of the administration of acids in cases of alkaline urine is not so powerful; but by means of carbonic and benzoic acids, acidity may be produced. Alkalinity of the freshly-passed urine may either be due to the presence of a fixed alkali\* (in which case it probably results

\* If the alkalinity be due to the presence of ammonia, the red litmus paper, which has been turned to blue by dipping in the urine, will regain its red tint after drying; but if the alkali be a fixed one, the blue tint will be permanent.



from some debilitating influence acting upon the system generally, or to the presence of ammonia. The latter form, which is by far the most common, points to some local disease in the bladder or urethra. Ammoniacal urine is frequently met with in cases of long-standing urethral stricture, chronic cystitis, spinal affections, &c. Highly acid urine, on the other hand, is met with in acute febrile diseases, and especially in acute rheumatism.

Normal urine undergoes, when kept too long, fermentative changes which, as they are liable to cause mistakes, must be carefully noted.

1. *Acid Fermentation*.—If the urine be allowed to stand exposed to the air, in a cool place, it will be found that its reaction increases in acidity steadily from day to day, and may continue to do so for as long as ten days. This fermentation is due to the presence of a peculiar organism, resembling yeast but smaller, and is accompanied by the precipitation of a yellowish-brown sediment, consisting of uric acid and urates, and frequently of oxalate of lime, along with clouds of mucus. The acidity is probably due to the formation of lactic and acetic acids.

2. *Alkaline Fermentation*.—After the acid reaction has fully developed itself, it gradually disappears, and the urine becomes alkaline. This change does not usually set in, when the urine is kept cool, before eight or ten days have passed, but if there be much pus or mucus present, the alkaline reaction may be detected much sooner; and if any admixture of old, decomposed urine be allowed to take place (as from the glass not having been thoroughly cleaned), it may come on in a few hours. The urine now becomes lighter in colour, opaque, and ammoniacal in odour, the urea having become changed into carbonate of ammonia. A white sediment separates, consisting of urate of ammonia, triple phosphate, amorphous phosphates, and carbonate of lime.

## CHAPTER XXV.

### Urinary System—(continued).

#### NORMAL CONSTITUENTS OF URINE.

THE normal constituents of the urine may be divided into two classes—organic and inorganic. Of these the following, which are the most important, will be here considered :—

##### *Organic Substances.*

1. Urea.
2. Uric Acid.
3. Creatinin.
4. Indican.

##### *Inorganic Substances.*

1. Chlorides.
2. Sulphates.
3. Phosphates.

**Urea** is by far the most important constituent of normal urine, and it is to be regarded as the chief product of the decomposition of albumen, and the last product of the regressive metamorphosis of the nitrogenous tissues of the body. The quantitative estimation of urea is therefore of much importance. It may be carried out in either of two ways.

(1.) *Estimation of Urea by means of Nitrate of Mercury.*—This method, which was first introduced by Liebig, depends upon the property which urea possesses of forming an insoluble compound with nitrate of mercury. When a dilute solution

of the mercury salt is added to a solution of urea, this precipitate forms so long as any urea remains unaltered. If, however, more nitrate of mercury be added when no more urea is present, a drop of the mixture when added to a solution of carbonate of soda gives a yellow precipitate of the hydrated oxide of mercury. In this way can be determined the exact point at which all the urea has been decomposed. Before this method can be applied, however, the phosphates and sulphates which the urine contains must be precipitated by means of a solution of baryta. For the volumetric analysis of urea we therefore require three solutions.

- 1 *A solution of nitrate of mercury*, of which 1 cub. centimetre corresponds to 0.01 gramme of urea. The method of preparation will be described in the Appendix.
2. *A solution of baryta*, prepared by mixing one volume of a cold saturated solution of nitrate of baryta with two volumes of cold saturated baryta water.
3. *A solution of carbonate of soda* of about twenty grains to the ounce.

The urine must first be freed from phosphates and sulphates, and for this purpose 40 c.c. are measured off by means of a pipette, and 20 c.c. of the baryta mixture added. After filtration, 15 c.c. of the filtrate (corresponding to 10 c.c. of the original urine) are measured off into a small beaker, which is placed under a graduated burette containing the standard solution of nitrate of mercury. From this burette small quantities of the solution are successively added to the urine, the mixture being all the time carefully stirred by means of a glass rod, and the additions are to be cautiously continued so long as a distinct precipitation follows each drop. When, however, the formation of the insoluble compound appears to be getting less distinct, and the analysis consequently approaches completion, a drop of the mixture must be removed on the

stirring rod and added to a small quantity of the solution of carbonate of soda, which has been placed on a porcelain plate, or better, in a watch-glass with a black back-ground. Should a white precipitate form, the addition of mercury must be continued, but as soon as a yellow colour appears when a drop is added to the soda solution, the analysis is at an end. The quantity of the nitrate of mercury solution which has been used is now to be read off, and a calculation made, remembering that every cubic centimetre corresponds to 0.01 gramme of urea. Corrections are, however, under certain circumstances necessary. If, in the course of the analysis, it be found that more than 30 c.c. of the mercury solution are being used for 15 c.c. of the urine mixture, we must add to the urine, before applying the carbonate of soda test, half the number of cubic centimetres of water as we have used of mercury solution above 30. Thus, if 48 c.c. of the standard solution have been dropped in, we should have to add 9 c.c. of water before we transferred a drop to the carbonate of soda solution. On the other hand, if less than 30 c.c. of the mercury solution have sufficed to precipitate all the urea, we must subtract from the total sum of the mercury solution used 0.1 c.c. for every 5 c.c. less than 30. For example, if only 20 c.c. of the standard solution have been used, we must subtract 0.2, leaving 19.8 as the sum from which we have to calculate. If great accuracy be required, the chlorides should be previously removed by precipitation with nitrate of silver. The modifications in this method which have been recently suggested by Pflüger\* are too complicated to be described here.

(2.) *The Estimation of Urea by means of Hypobromite of Soda.*—In principle this method was first described by Davy. It depends upon the fact that urea when treated with hypobromite of soda breaks up into nitrogen, water, and carbonic acid, the last of which is absorbed in the alkaline solution,

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\*Arch. f. d. ges. Physiol. xxi. and xxiii.

while the nitrogen comes off as free gas. Of the many forms of apparatus which have been described and are used for this analysis, perhaps the most simple is that of Dr. Graham Steele.\* It consists of an ordinary burette inverted in a tall glass cylinder containing water, and connected with a small conical glass vessel containing a short test-tube. After removing the test-tube, the conical vessel is filled to the depth of about an inch with the solution of hypobromite of soda (the method of preparing which will be presently described), and into this the test-tube is carefully slipped, after 5 c.c. of the urine to be tested have been placed in it. The cork of the conical vessel is then replaced, and the vessel dipped into water. The burette, which is now in communication with the conical vessel, is next raised or lowered as may be required, until the level of the water inside and outside is the same, and this point is read off. The conical vessel is now tilted over so as to allow the urine in the test-tube to flow out and to become mixed with the hypobromite solution. This mixture is followed by a rapid giving off of gas, and after all effervescence has ceased, and the nitrogen which has collected in the burette has had time to cool down to the temperature of the room, the burette is again moved so as to bring the water-level inside to the same height as that outside, and this point read off. The difference of the two readings gives the quantity of nitrogen which has been given off. Since we know that at the ordinary temperature of a room, 0.1 gramme of urea gives off 37.5 c.c. of nitrogen, the calculation is simple. This method, from the ease with which it can be carried out, is very convenient, but it is not extremely accurate, for not only urea, but also uric acid and creatinin, give off nitrogen when treated with hypobromite of soda. The error is, however, small. The preparation of the solution of hypobromite of soda is made as follows:—100 grammes of caustic soda are dissolved in water, and the solution diluted to 1250 c.c. To this 25 c.c. of bromine are to

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\**Edin. Med. Journal*, 1874.

be added, and the whole shaken vigorously. This solution must be kept in a stoppered bottle and in the dark. It decomposes rapidly, and can only be used when freshly prepared.

The quantity of urea excreted in twenty-four hours in healthy men averages from 300 to 500 grains. It is increased after exertion, and after a full meal of animal food. In almost all diseases attended with elevation of temperature, the urea elimination is increased. Thus in typhus, pneumonia, pleurisy, and acute rheumatism, the amount of urea excreted is usually much above normal. In diabetes mellitus, I have seen the daily quantity to be as high as 1800 grains. This is no doubt in part due to the large quantity of animal food consumed in this disease. On the other hand, the urea is diminished in almost all affections of the kidney, owing to defective eliminating power of that organ. Particularly is this the case with regard to acute inflammatory Bright's disease, and to the cirrhotic or contracting form, especially when in its later stages a degree of inflammatory action becomes superadded.

The urea excretion is affected by the administration of drugs, but much uncertainty exists as to the action of many of these. Phosphorus undoubtedly increases the elimination of urea, as do also most, if not all, diuretics. Morphia, quinine, and iodide of potassium, on the contrary, tend to diminish its quantity.

**Uric Acid** exists in normal urine in combination with potassium, sodium, ammonium, calcium, or magnesium; and, as all these salts of uric acid are very much more soluble in hot than in cold urine, they tend to separate out as the urine cools. The cloud of urates which thus so often appears soon after the urine is passed may be readily recognised by warming a small quantity of the urine containing the sediment in a test-tube, when it rapidly becomes clear. Uric acid is readily separated from urine by adding hydro-

chloric acid. It then deposits itself in crystalline form, the character of which will be described when we come to speak of urinary sediments.

*Detection of Uric Acid.*—It is often important to be able to detect the presence of uric acid in concretions and sediments. Very frequently this may be done by means of the microscope, but this is not always possible. Uric acid can, however, always be detected by means of the *murexid test*, which is applied as follows:—A small quantity of the sediment is dissolved in a porcelain dish with a few drops of nitric acid, and the solution so obtained is evaporated. To the reddish residue one or two drops of dilute ammonia are added, when the beautiful reddish-purple colour of murexid develops itself, which, on the further addition of a few drops of caustic potash, passes into bluish-purple.

The *quantitative estimation* of uric acid is difficult to carry out. A large quantity of urine (100-200 c.c.) is taken, and to it is added 5 c.c. of pure hydrochloric acid. The mixture is allowed to stand for forty-eight hours, and the precipitate of uric acid is then collected in a filter, washed with a little cold water, and weighed. Salkowski has however recently pointed out\* that by no means all the uric acid which the urine contains is thus removed by precipitation, and that the proportion which remains varies much in different urines. He recommends that the filtrate should be further treated with ammonia and magnesia, and after the precipitate of phosphates has been removed by rapid filtration, that the filtrate should be further treated with an ammoniacal silver solution. The precipitate which is thus formed, after careful washing, is then decomposed with sulphuretted hydrogen, the filtrate acidulated with hydrochloric acid, and the uric acid which then separates out collected and weighed.

The average quantity of uric acid excreted in twenty-four hours is about 0.5 to 1 gramme. It rises and falls simultaneously

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\* Virchow's *Archiv.*, vol. lii.

with urea, bearing the relation to that substance of 1 to 50 or 60 in the healthy state. It is much increased by an animal diet. In most feverish conditions the excretion of uric acid corresponds with that of urea, but when the respiration is interfered with, Bartels states that it is increased. During an attack of gout the excretion is diminished, but after the paroxysm is over, it undergoes some increase. It is increased in many hepatic affections, in leucocythæmia, in acute rheumatism, and sometimes in cases of simple indigestion. The appearance of a sediment of uric acid is only to be looked upon as pathological when it occurs either before the urine cools or immediately thereafter.

**Creatinin** is a normal constituent of urine, and is present in somewhat larger quantity than uric acid. Its presence is readily detected by adding to a small quantity of urine a few drops of a very dilute solution of nitro-prusside of sodium, when, on the further addition of dilute caustic soda a beautiful ruby red colour develops itself, which soon passes into deep straw yellow.

*Quantitative Analysis of Creatinin.*—300 c.c. of urine are taken, rendered alkaline by the addition of milk of lime, and then decomposed with chloride of lime until no more precipitation takes place. The filtrate is rapidly evaporated to the consistence of syrup, and mixed with 50 c.c. of alcohol (95 per cent.). The mixture is allowed to stand until all the chloride of sodium has separated out, is then filtered, and the filtrate evaporated down and treated with an alcoholic solution of chloride of zinc. After standing for three days, the zinc-creatinin chloride will have become fully separated, and may then be collected on a filter and weighed.

The quantity of creatinin excreted, which is normally about one gramme *per diem*, is increased in typhus and pneumonia and diminished in anæmia, chlorosis, and tuberculosis.

**Indican.**—It is not uncommon to encounter dark-yellow



urines, in which, on the addition of nitric acid, there becomes developed the dark-violet colour of indigo. The original pigment which is thus decomposed was first studied by Schunck,\* who took it to be identical with the indican of plants. Later investigations have however shown that the two substances are not quite identical, and that the indican found in urine is to be looked upon as potassium-indoxyl-sulphate. It is identical with the uroxanthin of Heller. Indican, when treated with a mineral acid, yields indigo, and this decomposition often takes place in the urine spontaneously after decomposition has set in, the indigo appearing on the edge of the glass and on the surface of the urine as a glistening dark-blue film. Indican is a derivative from indol, which is a result of the changes which albumen undergoes in the intestines, and Jaffé was the first to point out,† what was subsequently confirmed by Baumann and others, that when indol was injected subcutaneously in animals, indican appeared in quantity in the urine.

*Detection of Indican.*—The original process of Jaffé, as modified by Senator,‡ is as follows :—In a large test-tube are placed 10-15 c.c. of the urine, and to this is added an equal quantity of fuming hydrochloric acid, and then a concentrated solution of chloride of lime drop by drop, until the blue colour is fully developed. The mixture is now to be shaken up with chloroform, after which the chloroform will sink to the bottom, more or less deeply tinged with indigo. By this means a rough idea of the quantity of indican present may also be arrived at.

The method of estimating accurately the quantity of indican as given by Jaffé§ is very complicated, and for its details the original must be consulted.

The quantity of indican contained in human urine averages

\* *Proceedings of the Royal Society*, 1857.

† *Centralbl. f. d. med., Wiss.* 1872.

‡ *Centralbl. f. d. med., Wiss.* 1877.

§ *Pfäuger's Archiv*, iii.

6.6 mgrm. in 1000 c.c. In disease the indican excretion has been frequently investigated, most fully perhaps by Hennige.\* The most important clinical point is that indican appears in large quantity in the urine when there is obstruction of the small intestine, while, when the obstruction is seated in the large intestine, no such augmentation takes place. In cases of ileus, therefore, when the exact seat of the disease is doubtful, the estimation of the indican in the urine may have considerable diagnostic value.

Passing now to the consideration of the inorganic substances which are found in normal urine, we come first to the

**Chlorides.**—The chlorine which is contained in urine exists in combination with potassium, sodium, ammonium, magnesium, or calcium. The presence of these chlorides may be detected by adding to a small quantity of urine in a test-tube a few drops of nitric acid, and then a small quantity of a solution of nitrate of silver. A white flocculent precipitate at once falls, consisting mainly of chloride of silver, but also containing combinations of silver with uric acid, creatinin, xanthin, and urinary pigments.

*Estimation of the Chlorides*—Mohr's method depends upon the fact that when to a neutral urine containing chloride and phosphate of sodium and a neutral salt of chromic acid a solution of nitrate of silver is added, there first occurs a precipitation of chloride of silver; and when the point is reached when all the chlorine contained in the chloride of sodium is so precipitated, there then begins the precipitation of the red chromate of silver. For this analysis we therefore require—

1. *A solution of nitrate of silver*, of which 1 c.c. corresponds to 10 milligrammes of chloride of sodium or to 6.065 milligrammes of chlorine.

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\* *Deutsches Archiv. f. kl. Med.*, xxiii. p. 271.

2. *A cold saturated solution of neutral chromate of potassium.*
3. *Pure nitrate of potassium.*
4. *Pure carbonate of lime.*

Since the presence of urinary pigments interferes with the accuracy of this method, the silver combining with them, they must first be got rid of in the following manner:—

10 c.c. of urine are mixed with 2 grammes of nitrate of potassium in a platinum capsule, evaporated to dryness, and finally heated in a naked flame until the carbon is completely oxidised. The residue is then dissolved in water in a beaker, acidulated with a dilute solution of pure nitric acid, and then neutralised with a little carbonate of lime. To the fluid so obtained, which need not be filtered, four or five drops of the chromate solution are added, and then the silver solution is gradually dropped into it from a burette, the mixture being constantly stirred. Reddish spots appear where the solution falls, but they disappear on stirring, so long as any chloride of sodium is present. So soon, however, as the whole of that salt is decomposed, the next drop of the silver solution gives rise to a permanent red, which marks the conclusion of the operation. The amount of the silver solution which has been used is now read off, and as we know that each cubic centimetre corresponds to 6·065 milligrammes of chlorine, the calculation is easy.

The average quantity of chlorine excreted in the urine in twenty-four hours may be taken to be in the healthy state about 10 or 12 grammes. It is increased by the consumption of a greater quantity of common salt, and by the copious drinking of water.

In disease the most important change which occurs in the elimination of the chlorides is the remarkable diminution met with in acute feverish conditions, particularly in pneumonia. As the result of a very interesting series of observations, Röhmann\* comes to the conclusion that this diminution is due

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\* *Zeitschrift f. kl. Med.*, vol. i. p. 513.

not to a decrease in the amount of chlorine taken along with the food, but to a change in the relation of the albumen of the blood to the chloride of sodium in the plasma.

**Sulphates.**—The sulphates which are found in the urine are derived from the breaking up of albumen, either that of the tissues or that which is contained in the food. Sulphuric acid exists in the urine in two forms—first, in combination with the alkalies; and, second, as Baumann has shown, in the form of aromatic ether-sulphuric acid—chiefly phenyl-sulphuric acid and indoxyl-sulphuric acid. Both of these aromatic bodies when heated with hydrochloric acid break up into phenol or indigo or sulphuric acid. Acetic acid does not cause this decomposition.

*Detection of the Sulphates.*—Acidulate strongly with acetic acid, and on the addition of chloride of barium a white precipitate of sulphate of barium will fall, representing the sulphuric acid which was combined with the alkalies. If now the mixture be filtered and heated with hydrochloric acid, a further precipitate of sulphate of barium will fall, representing in this case the ether-sulphuric acid.

*Estimation of the Sulphates.*—If these two precipitates which have just been mentioned be weighed, the total amount of the sulphates, as well as that of each form, may be calculated. Fürbringer\* has pointed out an easy manner of obtaining results which are approximately accurate, and quite sufficiently so for comparative observations, which is to wash the precipitates in question into a very narrow graduated cylindrical vessel, and allow them to settle down. After some hours their upper level may be read off, and if the physician possess the result of only one weighing of such a precipitate, he can always translate into *weight* the height of his precipitates. For the purpose of such an analysis, however, at least 300 c.c. of urine must be taken.

The normal quantity of sulphuric acid which is excreted in

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\* Virchow's *Archiv.*, vol. lxxiii.

the urine in twenty-four hours is about two grammes. It is increased and diminished according as more or less albumen is broken up, and therefore it corresponds with the quantity of urea and uric acid, both in health and in disease.

Sulphur is also excreted in the urine in small quantity in the form of sulphy-cyanic acid, of taurin,\* and occasionally of cystin. Sulphuretted-hydrogen is also sometimes met with in the urine in disease.

**Phosphates.**—In normal acid urine phosphoric acid is met with in the form of the phosphates of the alkalies, sodium and potassium, and of calcium and magnesium. It may also appear in the form of glycerin-phosphoric acid and lecithin. It is derived in part from the food, and in part from the breaking down of tissues of the body which contain phosphorus, principally the osseous and the nervous structures.

When the urine loses its carbonic acid, as it does when heated, the earthy phosphates separate out as a white flocculent precipitate, which becomes redissolved on the addition of acid. The addition of ammonia to urine causes an amorphous precipitate of phosphate of lime, while the phosphate of magnesium unites with the ammonia to form ammonio-magnesian phosphate (triple-phosphate), which appears in a crystalline form. The microscopic appearance of all the various forms of phosphate will be described when we come to speak of urinary sediments.

*Estimation of Phosphoric Acid.* — The principle of Neubauer's method is the following:—When a hot solution of the phosphates in question is acidulated with acetic acid, it gives, with a solution of acetate of uranium, a precipitate of uranium phosphate. The point at which this reaction ends is, from the nature of the precipitate, difficult to determine, and it is consequently necessary to test the mixture from time to time with a solution of ferro-cyanide of potassium, which gives, when

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\* Salkowski-Virchow's *Archiv.*, vol. lviii.

there is present the slightest excess of the uranium solution, a dark reddish-brown coloration. The solutions required are:—

1. A solution of uranic oxide, of which 1 c.c. is equivalent to 0.005 gramme of phosphoric acid,  $P_2O_5$ .
2. A solution of acetate of soda prepared by dissolving 100 grammes of that salt in 900 c.c. of water, and adding 100 c.c. of concentrated acetic acid.
3. A solution of ferro-cyanide of potassium not too concentrated.

To 50 c.c. of the urine are added 5 c.c. of soda solution, and the mixture is placed in a beaker glass, and warmed in a sand-bath. From a burette the uranium solution is gradually added to the urine, until no further precipitation appears to take place. A drop is now removed, placed on a porcelain slab, and mixed with a drop of the solution of ferro-cyanide of potassium. If there be any excess of uranium—*i.e.*, if the analysis be at an end—a reddish-brown precipitate will appear where the drops come in contact. If this reaction does not take place, more uranium solution must be added to the urine. Each cubic centimetre of the uranium solution used corresponds to 0.005 gramme of phosphoric acid, so that the calculation is easy.

If it is wished to estimate separately the earthy phosphates, these must be precipitated by the addition of ammonia, the precipitate carefully separated by filtration, dissolved in water with the addition of a little acetic acid, and the solution treated in the manner just described.

The average quantity of phosphoric acid which is excreted in the urine in twenty-four hours is in the adult about three grammes, two-thirds of which may be taken to consist of the phosphates of the alkalies, and one-third of earthy phosphates. The quantity depends to a large extent upon the food—animal diet giving rise to more excretion than vegetable—and upon the condition of the alvine secretion, the earthy phosphates in particular being much increased in quantity when there is much constipation. Tissue change also influences the phos-

phatic elimination to a large extent, chiefly that which takes place in the nervous structures.

In the feverish state the phosphates are at first diminished, but when convalescence sets in their amount in the urine is increased to a point above normal. In chronic nervous diseases the phosphates are usually present to an excessive amount in the urine, and in osteomalacia the earthy phosphates are increased to such a degree that they may be found to be in excess of the phosphates of the alkalies. In meningitis also the phosphates have been found to be increased, and this fact is often useful in the differential diagnosis of that complaint.

## CHAPTER XXVI.

### Urinary System—(*continued*).

#### ABNORMAL CONSTITUENTS OF URINE.

THE substances which are commonly grouped under this heading are some of them present in normal urine, as, for example, sugar and oxalic acid; but their quantity is then so small as to elude detection by the ordinary methods of analysis, and it is only under pathological conditions that they appear in sufficient quantity to require notice. Other members of this group are, however, never present in normal urine.

**Albumen.**—The presence of albumen in the urine is one of the most important diagnostic indications which the physician can encounter. The special circumstances under which albuminuria occurs will be detailed presently. In the meantime we have to consider the methods by means of which its presence may be detected, and its quantity estimated.

The chief albuminous substances which appear in the urine are—

1. Serum-albumen.
2. Serum-globulin (paraglobulin).
3. Propepton.
4. Fibrin.

The first two of these substances are both detected by the ordinary tests of albumen. Their separation will be subsequently considered.



**Detection of Albumen** (serum-albumen and serum-globulin).

--Before testing for albumen, the urine in question must, if not already clear, be rendered so by careful filtration. Of the many methods employed, the following are the most important:—

(1.) *Boiling Test*.—If a small quantity of urine be placed in a test-tube, and heated in the flame of a spirit-lamp or Bunsen burner, it will be found that when the temperature has risen to near the boiling-point the albumen, if present, separates out as a white cloud, which, on standing, collects at the bottom of the tube in fine flakes. If the urine contains much earthy phosphates, these are apt to separate when the tube is heated, and the cloud so formed may be mistaken for albumen. It is, however, dissolved on the addition of a few drops of acetic acid. If the urine be alkaline to begin with, the albumen may not be separated out on boiling. It is therefore necessary to acidulate with a few drops of acetic acid; but inasmuch as there is some risk of adding too much of this acid, and so preventing the albumen reaction from taking place, it is best to proceed in all cases as follows:—

5-10 c.c. of urine are placed in a test-tube acidulated with acetic acid, and 1-6 of its volume of concentrated solution of sulphate of magnesia added. If albumen be present, there will now appear on heating a more or less distinct cloudiness. This test is absolutely trustworthy, and very delicate.

(2.) *Nitric Acid Test*.—A small conical glass is taken and filled about one-third full of urine. Down its side, while it is held inclined, are poured slowly a few drops of strong nitric acid, in such a way that when the glass is again held in an upright position, the acid forms a distinct layer at the bottom. If albumen be present in the urine, a cloud will form at the line of junction of the two fluids.

If the urine contain a large quantity of neutral urate of sodium or ammonium, the addition of nitric acid may cause the separation of the acid urates in the form of a cloud. This cloud lies near the upper surface of the urine, and is therefore not

readily mistaken for albumen ; but in cases of doubt it is only necessary to warm the glass, and so cause solution of the cloud, or to dilute the urine previously with twice or thrice its volume of water, after which no such cloud will form.

(3.) *The Ferro-cyanide Test.*—To the urine contained in a test-tube a drop or two of acetic acid is to be added, and then a small quantity of a solution of ferro-cyanide of potassium. If albumen be present, a white flocculent precipitate will separate out in the cold. The ferro-cyanic test pellets introduced by Dr. Pavy, are very convenient for bedside urine testings, as no addition of acid nor heating is required in their use.\*

Other tests, such as those in which carbolic, tannic, and metaphosphoric acids are employed, appear to be wholly unnecessary.

**Estimation of Albumen.**—It is often of great importance to the physician to know the quantity of albumen which is being excreted in the urine from day to day. Unfortunately there is no very ready method of performing such an analysis. The most accurate is that of Berzelius. The urine is carefully filtered—10-15 c.c. of the filtrate placed in a porcelain dish, carefully acidulated with acetic acid and evaporated to dryness on a water-bath. The remainder is extracted first with hot water, and then with alcohol, placed upon a weighed filter, dried at 100° C., and finally weighed. From the result so obtained must be subtracted the quantity of earthy phosphates and colouring matter which the residue contains, and this is done by burning the filter and the coagulum in a platinum capsule, and deducting the weight of the ash so obtained.

The quantity of albumen present in urine may likewise be estimated by means of Laurent's polarimeter. The urine must be rendered very clear by means of filtration, or, if this fail,

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\* Dr. Oliver of Harrogate has recently suggested the use, for clinical work, of small pieces of filter-paper saturated with various of the salts which precipitate albumen.

by the addition of a little milk of lime and subsequent filtration. If the tube 1 decimeter long be used, then each degree of polarisation to the left corresponds to 1 gramme of albumen in 100 c.c. of urine. This method of estimating albumen is, however, unfortunately not very accurate.

*Serum-globulin* (paraglobulin) may be readily detected in urine by means of Hammarsten's method.\* If the urine be saturated with magnesium sulphate, which is to be added in the form of a fine powder, the globulin will separate out as a white flocculent precipitate. According to Estelle,† the best method for estimating separately the serum-albumen and the serum-globulin is as follows:—He takes a small quantity of urine and adds to it sulphate of magnesia until no more will dissolve. The mixture, after having been shaken for ten minutes, is poured upon a weighed filter, the precipitate washed with hot water, and finally with distilled water, until the sulphate of magnesium is completely removed, as indicated by the fact that chloride of barium when added to the washings gives no longer a precipitate. The filter containing the precipitate is then dried and weighed. In this way the quantity of serum-globulin is obtained. The serum-albumen which was contained in the original quantity of urine is now contained in the first filtrate, and its amount can be estimated by coagulating with acetic acid, and boiling, filtering, and weighing in the manner already described. The clinical bearings of serum-globulin will be presently referred to.

*Propepton*.—The peculiar albuminous substance, which Bence Jones first described ‡ as occurring in the urine of a patient suffering from mollities ossium, is in all probability identical with Schmidt-Mühlheim's propepton and with Kühne's hemi-albumose. It is an intermediate product of the digestion of

\* Pflüger's *Archiv.*, vols. xvii. and xxii.

† *Revue Mensuelle*, 1880, p. 704.

‡ *Phil. Trans.*, 1848.

pepsin or trypsin before pepton is formed. Virchow found \* it to be present in the medullary substance of the bones in cases of mollities ossium, Lassar detected it † in the urine of petroleum poisoning, and Neale ‡ in a case of hæmoglobinuria. Its chief peculiarity is that though precipitated by nitric acid in the cold it becomes dissolved on heating, and again separates out when the mixture is allowed to cool.

*Detection.*—The urine must first of all be freed from serum-albumen and serum-globulin. If the urine be acidulated with acetic acid, saturated with sulphate of magnesia, heated to boiling, and then filtered hot, these two substances will remain on the filter while the propepton will pass through in solution in the hot filtrate, and will separate out as cooling takes place.

*Fibrin*, when present in the urine, is usually in the form of flakes, and is due to inflammatory action in the kidneys and urinary passages. It is probable that the casts of the renal tubules, of which we shall presently come to speak, are chiefly composed of fibrin. The spontaneous coagulation of chylous urine is due to the presence of fibrin.

The transitory occurrence of albumen in the urine has been frequently observed in persons who are apparently in good health. Such cases have been described by Ulzmann, § Fürbringer, ¶ Bamberger, || Rüneberg, \*\* and others. Apart from such intermittent albuminuria, the chief conditions which give rise to albuminuria of a more or less permanent character may be grouped as follows:—

(1.) In most febrile conditions albumen may appear in the urine, but usually only in small quantity. When the amount

\* Virchow's *Archiv.*, iv.

† Virchow's *Archiv.*, lxxvii.

‡ *Lancet*, 1879.

§ *Wiener med. Presse*, 1870.

¶ *Deutsch Archiv. für kl. Med.*, vol. xxvii.

|| *Wiener med. Wochenschr.*, 1881.

\*\* *Deutsch Archiv. für kl. Med.*, vol. xxvi.

is considerable, it points to the occurrence of Bright's disease as a complication.

(2.) In those affections of the heart or lungs which lead to circulatory changes in the kidneys, in particular to venous engorgement, albumen appears in the urine ; but here, also, the quantity is not very great.

(3.) In Bright's disease. In all the different forms of this affection albumen appears in the urine. Its amount is greatest in the inflammatory form (in severe cases the urine may even become solid on heating), and ~~whenever we~~ find a large quantity of albumen, the presence of this form of Bright's disease must be suspected. In the cirrhotic and waxy forms, when these are uncomplicated with inflammation, the albumen is usually only present in very small amount ; indeed, albuminuria often does not show itself until some considerable time after the commencement of the morbid process in both of these disorders, and when it does set in, it is often subject to distinct remissions, appearing one day and disappearing the next. It is of great importance to distinguish the albuminuria of Bright's disease from that which occurs in other disorders, and particularly from that of heart disease ; and while a consideration of the whole circumstances of the case will generally lead to a correct diagnosis, the most important point is the presence of renal tube-casts and epithelium, which are usually more or less abundant in Bright's disease.

(4.) Various nervous disorders are accompanied with albuminuria. It occurs, for example, after an epileptic fit ; and Warburton Begbie was the first to point out\* that albumen was frequently to be found in the urine in cases of exophthalmic goître.

Albuminuria also occurs in cases of lead poisoning, and sometimes in pregnancy.

In regard to the occurrence of serum-globulin, little of diagnostic importance is known. Senator states that while it is

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\* *Edin. Med. Journal*, 1874.

almost always present in albuminous urine, it is most abundant in the waxy form of Bright's disease. In the article to which I have already referred, Estelle states that it is invariably to be found in albuminuria: that it is often present in greater quantity than the serum-albumen; and that sometimes it exists alone.

The albumen found in the urine may in certain cases be derived from blood, pus, or spermatic fluid with which the urine has become mixed.

**Pepton.**—Closely allied to these albuminous substances stands pepton, which Gerhardt was the first to describe as occurring in urine. The various peptons, which are the products of the digestive action of pepsin and pancreatic juice upon albumen, are not precipitated by acetic acid and ferro-cyanide of potassium (as all other albuminous substances are), but are thrown down by tannic acid, phosphor-wolframic acid, and certain other reagents. Peptons further give the biuret reaction (purple-red) with sulphate of copper and caustic soda, and also a red colour with Millon's reagent.

To detect pepton in albuminous urine the albumen must be completely separated by means of acetic acid, boiling, and filtration, and then by the addition of hydrated oxide of lead, the lead being subsequently removed by means of sulphuretted hydrogen. This process must be repeated until no trace of albumen can be detected by the ferro-cyanide test. The pepton in the filtrate is then precipitated by means of a solution of tannin, the precipitate collected and washed, the tannin removed by means of the hydrate of baryta, and the baryta by means of dilute sulphuric acid. Finally, the clear filtrate which is thus obtained is tested with Millon's reagent, when a red colour will show the presence of pepton.

The subject of peptonuria has been chiefly investigated by Maixner\* and Hofmeister.† According to the former obser-

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\* *Prager Vierteljahrschrift*, cxliii. (1879), p. 78.

† *Zeitschrift für phys. Chemie.*, iv. (1880), p. 253.

ver, pepton appears in the urine very frequently in those diseases in which the formation and collection of pus play a prominent part,—such as purulent effusions into the pleural and peritoneal cavities, abscesses in various situations, pyonephrosis, bronchorrhœa, and phthisis when cavities have formed. Peptonuria also occurs, according to the same author, in the stage of resolution of croupous pneumonia, in phosphorus poisoning (confirmed by the observations of Schultzen and Riess), in typhoid fever, and in carcinoma of the stomach.

The appearance of pepton in the urine is to be explained on the supposition that when it passes into the blood it does not undergo the usual change, and so reaching the kidneys as pepton, it is excreted as such. Ploz and Gyergyai\* have shown that when peptons are injected into the blood-stream, provided that they are not thrown into the portal circulation, they appear unchanged in the urine.

**Mucus.**—A small quantity of mucus is present in normal urine; but in such affections as catarrh of the bladder or urethra it may be much increased. It is sometimes of importance to be able to distinguish mucus from pus in the urine. This is readily done by filtration, when if the pus be present, the filtrate will give the reactions of albumen; but if it contain mucus it will give that which is characteristic of mucin—i.e., when acidulated with acetic acid a precipitate of mucin separates out in the cold.

**Sugar.**—Even in normal urine a small quantity of grape sugar is present; but its quantity is so minute as not to give evidence of its presence with the ordinary tests which are about to be described. When, therefore, sugar is detected by their means, it is present in abnormal amount, and constitutes the pathological condition termed glycosuria.

\* Pflüger's *Archiv.*, vol. x.

*Qualitative Tests for Sugar.*

The qualitative tests for urine containing sugar depend upon the coloration caused by boiling with caustic potash, upon the power grape sugar possesses of reducing hydrated oxide of copper, and upon the evolution of carbonic acid when fermentation is set up by the addition of yeast. In all cases albumen, if present, should be got rid of by coagulation and filtration before these tests are applied.

1. *The Caustic Potash Test* (Moore's).—The urine is mixed in a test-tube with an equal quantity of liquor potassæ, and the upper part of the mixed fluid heated to boiling in the flame of a spirit-lamp. If sugar is present, the heated portion will assume a dark-brown colour. Almost all urines, it must be remembered, darken slightly when thus treated; but the change is very marked when sugar is present. This test is not very delicate, but is readily performed, and is useful as a preliminary.

2. *Trommer's Test*.—To a small quantity of urine in a test-tube,  $\frac{1}{3}$ rd of its volume of liquor potassæ is added, and then a drop or two of a solution of sulphate of copper. The precipitate which falls will redissolve (the more readily if sugar be present), and more of the copper solution must be added until a small quantity of the hydrated oxide remains as a precipitate. On boiling this mixture a yellow colour will show itself if sugar be present, and will pass into a reddish-yellow granular precipitate of the suboxide of copper.

3. *Test with Fehling's Solution*.—Of all the tests for sugar this is by far the most delicate and satisfactory. The method of preparing Fehling's solution will be described on p. 277. A small quantity of that solution is placed in a test-tube heated to boiling, and then a drop or two of urine added. If sugar be present, reduction of the copper in Fehling's solution will at once take place, giving rise to a red precipitate. Fehling's solution is liable to undergo decomposition when kept for some time, and it will then of itself become reduced on boiling. If,



however, it be always boiled previous to the addition of the urine, no error can take place, for if the solution remain clear on boiling, it is in a fit state for use.

4. *Fermentation Test*.—Under the influence of yeast, grape sugar breaks up into alcohol and carbonic acid, and this evolution of carbonic acid has been made the basis of another qualitative test for the presence of sugar. It is most readily performed by taking two test-tubes or narrow phials, one filled with water and the other with urine, adding to each a small quantity of yeast, covering them with a saucer, and inverting them. If sugar be present in the urine, carbonic acid gas will collect at the upper part of that test-tube. A few bubbles of gas may come from the yeast itself, but the second test-tube containing water will show these also, so that any mistake is hardly possible. This test is not very sensitive. According to Roberts,\* urines containing two grains and a-half of sugar in the ounce, and under, yield no sign of sugar with this test.

*Quantitative Estimation of Sugar*.—A considerable number of methods have been devised for this purpose. I propose however, only to describe two—viz., first the modification of the process of Fehling which Dr. Pavy has recommended,† and second, the method by means of the polarimeter.

*Pavy's Method*.—The principle upon which Fehling's method for the volumetric analysis of sugar depends is the reducing action which that substance has upon hydrated oxide of copper; but the reaction is so much obscured by the red precipitate of the suboxide which is thrown down that the results are not very accurate. Pavy therefore devised the following method, in which ammonia is made use of to prevent the precipitation of the suboxide. If ammonia be added to Fehling's solution, and the mixture be boiled, a sufficiency of grape sugar may be added to the mixture to reduce all the copper and render the solution colourless, without any precipitation taking place.

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\* "Urinary and Renal Diseases," 4th ed., p. 188.

† *Proceedings of the Royal Society of London*, 1879.

The preparation of the copper solution is carried out as follows:—An ordinary Fehling's solution is made by dissolving 34·639 grammes of pure sulphate of copper in water and diluting to 500 c.c. The solution so obtained is mixed with another solution prepared by dissolving 173 grammes of tartrate of potassium and sodium in water, mixing it with 100 c.c. of liquor sodæ (sp. gr. 1·34), and diluting the mixture to 500 c.c. When these two solutions, each of 500 c.c., are united, we obtain one litre of ordinary Fehling's solution. Of this solution 120 c.c. are now taken, mixed with 300 c.c. of strong ammonia (sp. gr. ·880), and diluted up to a litre with distilled water. This constitutes Pavy's standard solution, and of it 20 c.c. correspond to 0·01 gramme of grape sugar.

The analysis is carried out as follows:—A flask of about 80 c.c. capacity is taken and fitted with a cork, through which two holes are bored, one of which receives the delivery tube of a Mohr's burette, and into the other is adapted a bent glass tube to allow of the escape of air and steam. The burette, filled with the urine,\* is fixed in its stand, and the flask, into which 20 c.c. of the copper solution have been measured allowed to hang free, so that nothing may obstruct the full view of its contents. Heat is now to be applied to the flask, and after the solution has boiled for a few minutes, so that all air has been expelled from the flask, the urine is allowed to flow into it until the copper solution has become completely colourless. This marks the end of the reaction. The quantity of urine used contains 0·01 gramme of grape sugar.

*Method by Circular Polarisation.*—Grape sugar when in solution possesses this peculiar property, that if a beam of polarised light pass through it the beam becomes rotated to the right, and the degree of this rotation is in exact proportion to the amount of sugar contained in solution, and the length of the column of solution which the light traverses. Several

\* It is best in the first instance to dilute the urine in the proportion of 10 to 100.

instruments have been devised for the purpose of measuring the degree of this right-handed rotation, and so estimating the quantity of grape sugar present. Of these, the best known is the saccharimeter of Soleil-Ventzke. Its construction is complicated, and I do not propose to describe it in detail. It consists of two short brass tubes lying in line, and containing various polarising prisms. Between these two end tubes fits in the tube containing the urine to be tested. By means of a milled head two quartz prisms are moved so as to compensate for the rotation effected by the sugar solution, and the amount of this movement is registered by means of an attached scale and vernier. When this scale stands at zero, and when no sugar solution is in the tube, the appearance presented on looking through the instrument is a circular field divided into two lateral halves, each of which presents the same tint. If now the tube containing diabetic urine be slipped into its place, the light becomes rotated, and, on account of the special arrangements of the instrument, the field of vision assumes a different colour on the two sides. By slowly moving the screw which commands the quartz prisms, these two colours become gradually altered in tint until they again exactly correspond to each other. The amount of movement required to effect this is now to be read off on the scale by means of the vernier, and by a simple calculation we can learn the percentage of sugar in the urine in question. With a tube one decimetre long each degree of the scale represents 1 gramme of grape sugar in 100 c.c. of urine.

The urine must always be rendered perfectly clear by means of filtration before it is placed in the tube of the saccharimeter, and if it is highly coloured it is well to remove the pigment by precipitation with acetate of lead and filtration. Albumen rotates polarised light to the left, as has been already mentioned, hence it is absolutely necessary to get rid of this substance, if it be present, before the saccharimeter is used.

Laurent's polarimeter is more accurate in its readings, and is to be preferred. The calculation is the same as that of the Soleil-Ventzke saccharimeter.

Saccharine urine is rarely met with except in cases of diabetes mellitus. Diabetic urine possesses, when the disease is fully developed, various well-marked characteristics. It is large in quantity, sometimes reaching so high a figure as 15 or 16 pints, and correspondingly pale, but nevertheless possesses a high specific gravity, ranging from 1040 to 1050, or even higher. The quantity of the nitrogenous substances excreted is usually, if not invariably, very much increased. The quantity of grape sugar excreted may, in severe cases, be as high as 25 or 30 ounces in twenty-four hours.

In certain cases of diabetes there may be detected in the urine, towards the termination of the case, a peculiar etherial odour, the result of the formation of *aceton*; and to the presence of this substance in the blood have been, by some, ascribed the symptoms of diabetic coma. Such urine usually assumes a reddish-brown colour on the addition of chloride of iron, and this reaction is supposed to be due to the presence of ethyldiacetic acid, which, as Geuther and Gerhardt have pointed out, readily breaks up into aceton, alcohol, and carbonic acid. Much uncertainty, however, involves this interesting point.

**Blood** may be found in the urine as such (hematuria), or only blood pigment may be present (hæmoglobinuria); and these two conditions are readily distinguished by the fact that in the former case blood corpuscles are found on microscopic examination, while in the latter they are absent. The admixture of even a very small quantity of blood gives the urine a peculiar smoky appearance. When it is present in larger quantity the urine become bright-red or dark-brown. Small quantities of blood are best detected by means of the microscope, but when no corpuscles or crystals of hæmatin are present, recourse may be had to the spectroscope. If oxy-hæmoglobin be present, two dark absorption bands will be seen lying between the lines D and E. On the addition of sulphide of ammonium to the specimen of the urine the spectrum of reduced hæmoglobin will appear—a broad dark band

also lying between D and E, and less well defined than the bands of oxyhæmoglobin.

In cases of hæmaturia it is important to ascertain from what point in the urinary tract the blood comes, and this is not usually difficult. The hæmorrhage may come :—

(1.) *From the Urethra*.—The blood is mixed with the first portion of urine passed, often being expelled as a long clot, and it continues to flow in the intervals of micturition.

(2.) *From the Neck of the Bladder*, or prostatic part of the urethra.—In this case the blood usually appears only at the very end of micturition, when the sphincter vesicæ begins to contract.

(3.) *From the Bladder*.—The blood is usually coagulated, and is passed in clots as large as the calibre of the urethra will allow to escape.

(4.) *From the Ureters*.—In this case the blood often appears in the form of long worm-like clots, which are casts of the ureters.

(5.) *From the Kidneys*.—When the blood comes from the kidneys it is uniformly diffused through the urine, is almost never in very large quantity, and when the urinary sediment is examined there are found tube-casts, usually containing blood corpuscles.

*Hæmoglobinuria* appears in such diseases as purpura, scurvy, pyæmia, severe typhus, small-pox, &c., and results from a breaking down of the red blood corpuscles in the blood stream, and the consequent liberation of the hæmoglobin they contain, which then escapes into the urine. It also occurs in a paroxysmal form, each paroxysm being accompanied with ague-like symptoms, and is then usually the result of a chill, although sometimes it may be traced to malarial infection.

**Bile Pigment** appears in the urine in cases of jaundice. Its presence is readily detected by the play of colours which ensues when the urine which contains the pigment comes in contact with nitric acid. If a little urine containing bile pigment is

placed in a conical glass, and a few drops of nitric acid (which has been allowed to stand exposed to light for some time, and is therefore mixed with nitrous acid) are allowed to run down the edge of the glass and collect at the bottom of the vessel, a series of coloured rings will form in the following order: yellow, violet, blue, and green. A still better method is to filter the urine and then to allow a drop of nitric acid to fall on the surface of the filter. The play of colours ending in green will then be very distinctly seen.

Urine containing bile pigment is usually yellowish or greenish. It froths easily, and the bubbles of foam have a greenish-yellow colour.

**Bile Acids** are found in the urine in considerable quantity in hepatogenic icterus. When to a solution of these acids a little cane sugar is added, and then a drop or two of sulphuric acid, a beautiful purple colour develops itself. On this reaction depends their detection in urine. In a small quantity of urine a little sugar is dissolved, a strip of filter-paper is dipped in, and then allowed to dry. If now a drop of sulphuric acid be allowed to fall upon the paper, a purple ring will appear round it if bile acids are present.

## CHAPTER XXVII.

### Urinary System—(*continued*).

#### URINARY SEDIMENTS.

IN order to examine the sediment of a urine it is best to allow the urine to stand covered for some hours in a conical glass, after which a drop or two of the sediment which has collected may be removed by means of a pipette and examined microscopically. It is of considerable importance to ascertain the reaction of the urine at the time of its depositing, and to note whether the specimen has been freshly passed or not.

Urinary deposits are divided into two classes—organic and inorganic. Of these the first is by far the most important.

#### *Organic Deposits.*

These include blood and pus corpuscles, epithelium, tubercasts, spermatozoa, and micro-organisms.

1. **Blood Corpuscles** are found in the urine in cases of hæmaturia (see p. 279). When the urine is acid the corpuscles preserve for some time their normal appearance; but when it is alkaline, or very dilute, the red corpuscles swell up, lose their biconcave shape, and become pale. On the other hand, when the urine is concentrated they shrink up and become crenated. It is very rare to find rouleaux of corpuscles. They are only seen in cases of profuse bleeding from the bladder. Occasionally crystals of hæmatoidin may be found.

**2. Pus Corpuscles** when present in any quantity form a yellowish-white deposit, which is usually easily recognisable by the naked eye. Microscopically, the corpuscles present as a rule their normal appearance; but if the urine be strongly alkaline, they tend to run together and form a homogeneous mass. If there be doubt as to whether a deposit consists of pus it is only necessary to add a small piece of caustic potash and to stir with a glass rod, when, if the sediment be formed of pus, it will become tenacious, glassy, and semi-solid.

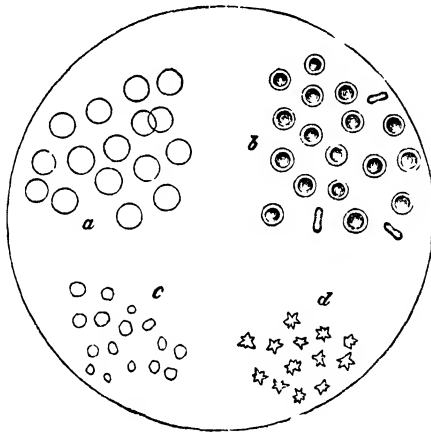


FIG. 33 —Blood Corpuscles in Urine. (Roberts.)

(a) Swollen up.

(b.) Showing biconcave shape.

(c.) Shrunken.

(d.) Crinkled

The presence of pus in the urine is always a sure sign that there exists an acute or chronic inflammation at some part of the urinary tract—renal abscess, pyelitis, cystitis, or urethritis. In women it must be remembered, pus flowing from the genital tract may become mixed with the urine.

**3. Epithelium.**—The epithelial cells in the urine are best seen when stained with eosin or fuchsin. They may be derived from any portion of the urinary tract. The epithelium of the urinary tubules consists of round or polygonal cells, each



having a large and sharply defined nucleus. Those of the pelvis of the kidney are conical, with one, or sometimes two, tail-like processes. The large irregular pavement epithelial cells which are often seen in the urine come from the bladder or vagina.

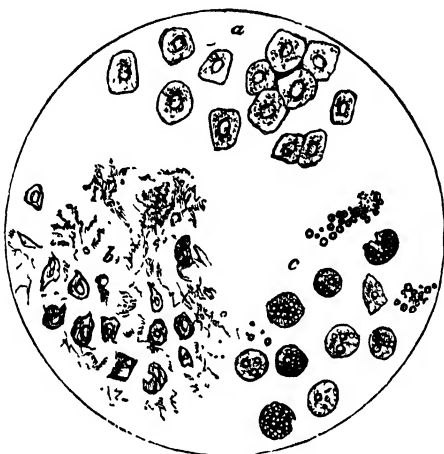


FIG 34.—Renal Epithelium in Urine (Roberts )  
 (a ) Natural appearance. | (b ) Atrophied and Disintegrated.  
 (c ) Fatty Degeneration.

**4. Renal Tube-casts.**—Before examining the urine for tube-casts, a specimen should be allowed to stand in a conical glass for twenty-four hours, at the end of which time a few drops of the sediment may be raised by means of a pipette and examined microscopically (*vide* fig. 35). Staining with eosin or methyl-green will make the tube-casts more distinct.

Renal tube-casts\* are almost invariably associated with albuminuria, and most frequently with Bright's disease, but they occasionally occur when no albumen can be detected in the urine. They are, as a rule, formed of fibrin, and are, as

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\* See an article by Dr. George Johnson on the various forms of renal tube-casts.—*British Medical Journal*, March, 1882.

their name implies, casts of the renal tubules, in the majority of cases of the convoluted tubules of the cortex. The chief forms of tube-casts are the following:—

(1.) *Epithelial Casts*.—In these the fibrinous cylinder has become covered over with epithelial cells which have been detached from the lining membrane of the tubule. These cells

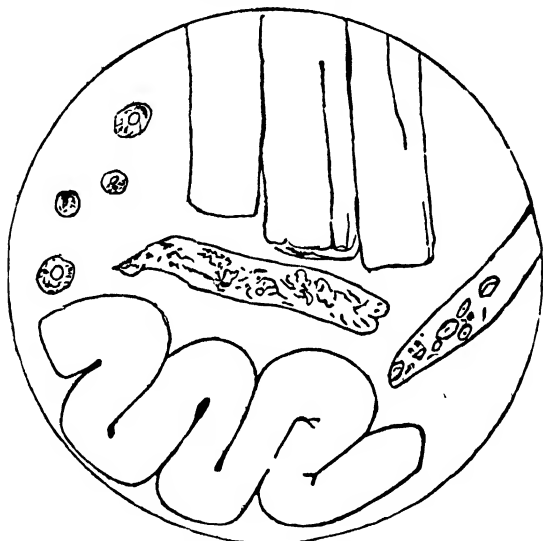


FIG. 35.—Renal Tube Casts. (Roberts.)

may be more or less cloudy and swollen. Such tube-casts are found in the inflammatory form of Bright's disease.

(2.) *Pus Casts*.—Casts containing pus corpuscles embedded in them are sometimes met with. According to Dr. George Johnson, they are diagnostic of glomerulo-nephritis.

(3.) *Fatty Casts*.—Very frequently casts are found studded over with oil globules. These globules are the result of fatty degeneration of the renal epithelium, and such casts are met with in the second stage of inflammatory Bright's disease.

(4.) *Granular Casts*.—Dark opaque granular casts are also the result of epithelial degeneration in the renal tubules.

(5.) *Blood Casts* may either consist wholly of blood, the corpuscles being closely applied to one another, or fibrinous casts may be seen containing one or two blood corpuscles embedded in them. Such casts point to capillary rupture, and are found in acute inflammatory Bright's disease.

(6.) *Hyaline Casts* are clear, homogeneous, and transparent, sometimes so delicate in structure as to be barely visible. They are for the most part formed in the convoluted tubules of the cortex, and have therefore a correspondingly convoluted form. The smaller specimens have been moulded within the lumen of a tubule which still retains its epithelium, while larger varieties have been formed in tubules previously denuded of epithelium, and therefore of greater capacity. Hyaline casts point to chronic Bright's disease.

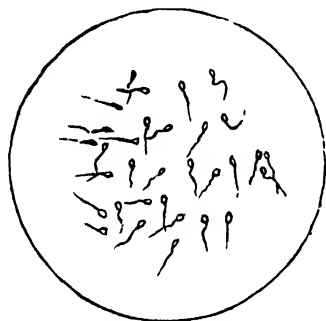


FIG. 36.—Spermatozoa. (Roberts.)

Occasionally hyaline casts may be found which exhibit the amyloid reaction, becoming reddish-brown on the addition of iodine, and dirty violet on the further addition of sulphuric acid; and giving also a beautiful violet with methyl-green, which tinges other casts green. Such waxy or amyloid casts are more strongly refractive than

the ordinary hyaline variety, and being less flexible they exhibit deep fissures where they have been torn asunder in passing through the straight tubules.

The student should be careful not to mistake for tube-casts these mucus-coagula which are so often found enclosing in their meshes whatever amorphous inorganic deposit the urine may happen to contain.

**5. Spermatozoa** are occasionally found in urine. They preserve their normal appearance for a long time. If the

urine be very fresh, they may even be seen in active motion, but these movements are soon lost. Urine which contains spermatozoa becomes alkaline very rapidly.

**6. Micro-Organisms.**—Very many forms of lower organisms are found in urine. Many of these only develop after the urine has been voided, and are derived from the atmosphere. Occasionally, however, urine as it leaves the body will be found to contain such organisms. These may be the result of the introduction of a catheter which has not been properly purified, but in other cases they are undoubtedly derived from the blood. The latter are most frequent in acute infectious disorder.\* Roberts has described† a peculiar



FIG. 37.—Sarcinae. (Roberts)



FIG. 38.—Embryo form of Bilharzia in Urine. (Roberts.)

condition which he calls bacilluria, in which the urine when

\* Kannenberg, *Zeitschr. f. kl. Med.*, i. p. 506.

† *Trans. of the International Medical Congress*, 1881.

passed is opalescent from the presence of enormous numbers of bacilli. The reaction is acid and when the urine has stood for some time the organisms sink to the bottom of the glass, leaving the supernatant fluid clear.

Occasionally the embryo forms of parasites, as for example the *Bilharzia hæmatobia*, which infest the blood, are found in the urine.

### *Inorganic Sediments.*

The reaction of the urine in which the sediment is found gives an important indication as to its constitution, certain substances separating out only in acid urine, while others are only found when the reaction is alkaline. The following table shows what the physician may be prepared to meet with in each case :—

<i>Acid Urine.</i>	<i>Alkaline Urine.</i>
Amorphous—	Amorphous—
Urates of potash and soda.	(a.) Neutral phosphate of lime.
	(b.) Carbonate of lime.
Crystalline—	Crystalline—
(a.) Uric acid.	(a.) Urate of ammonium.
(b.) Oxalate of lime.	(b.) Crystallised phosphate of lime.
(c.) Leucin.	(c.) Phosphate of magnesium.
(d.) Tyrosin.	(d.) Phosphate of ammonium and magnesium (triple-phosphate).
(e.) Cholesterin.	
(f.) Cystin.	

### **Sediments of Acid Urine.**

1. *Urates*.—The amorphous deposit of urates, which is so frequently met with even in healthy urine, consists in the main of urate of soda, but may also contain urate of potash and of magnesia. To the naked eye the deposit of amorphous urates has a reddish, brick-dust colour, due to pigmentation with uroerythrin. When the urine has been allowed to stand in a glass for some time, and deposit these urates, a

peculiar bloom may be seen upon the sides of the glass when it is inclined, which is a characteristic and unmistakable sign of the presence of urates. Microscopically this deposit appears amorphous and finely granular. On warming the microscope slide the sediment becomes dissolved, and it separates out again on cooling, and the same reaction can be very readily seen with a larger quantity in a test-tube.

In health a deposit of urates often occurs after profuse sweating and violent exercise, in cold weather. Pathologically this deposit is found in all febrile conditions, in grave organic disease, particularly of the liver, and in dyspepsia. Very rarely a crystalline deposit of urate of soda may be found, in the form of irregular masses with spiny projec-



FIG. 39.—Urate of Soda. (Roberts.)



FIG. 40.—Uric Acid, simpler forms. (Roberts.)

2. *Uric Acid*.—In Chapter XXV. the more important points

connected with the elimination of uric acid have been stated. There only now remain for consideration the appearances which a deposit of this substance presents in the urine.

The crystals of uric acid can usually be seen as bright reddish-brown grains adhering to the sides of the glass, or forming a layer at the bottom. They closely resemble the grains of cayenne pepper. Microscopically these crystals vary much in shape. They may take the form of four-sided tables, of six-sided rhombs, or they may be lozenge-shaped, ovoid, or barrel-shaped, or still more elongated and arranged in a stellate



FIG. 41.—Uric Acid, stars and spikes. (Roberts.)

fashion. In whatever form uric acid appears the crystals are always more or less yellow; and as no other crystal which spontaneously separates out from urine is so tinted, there can be no difficulty in its recognition.

3. *Oxalate of Lime* appears in the urine as small octahedra, which may be more or less elongated, or in the form of dumbbells or small ovoids. To the naked eye the deposit appears as a white, undulating, clearly-defined layer, resting upon a greyer deposit beneath.

Oxalic acid occurs normally in urine in minute quantities, and is much increased by the consumption of sugar, and of such vegetables as contain it in their tissues. It constitutes one of the last stages in the decomposition of the effete tissues, and probably results from impeded metamorphosis (Beneke). Oxaluria is frequently produced by conditions which interfere with respiration and circulation. As regards the symptoms of oxaluria, there is much difference of opinion, but it appears certain that in many cases at all events the appearance of oxalate of lime in quantity in the urine is accompanied with a well-marked train of symptoms. The patient is usually emaciated, very nervous, extremely hypochondriacal, and irritable.

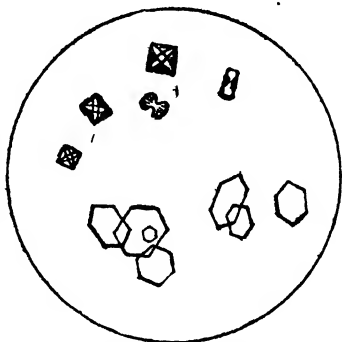


FIG. 42.—Oxalate of Lime and Cystin.

There is pain across the small of the back, a degree of irritability of the bladder, and general muscular weakness. It must be remembered, however, that, on the one hand, such a train of symptoms may be present in full development without any oxaluria, and, on the other, that crystals of oxalate of lime may be very abundant without producing any such symptoms.

4. *Leucin and Tyrosin*.—Leucin appears in the urine microscopically in the form of larger or smaller yellowish-brown balls, which sometimes show distinct striation. Tyrosin, on the other hand, appears under the microscope as sheaves of silky glittering needle-shaped crystals. These two substances result from the decomposition of albumen and other nitrogenous bodies, and when they appear in the urine they are a proof of incomplete oxidation of these substances, and they therefore precede urea. Leucin and tyrosin are very rarely met with in urinary deposits. Their occurrence is almost con-



finer to cases of acute yellow atrophy of the liver, though they are not by any means invariably met with in that disease.

5. *Cholesterin* and other fats are found in the urine in cases of chyluria. The deposit consists of minute oil globules, and when dissolved in a mixture of alcohol and ether, and the solution allowed to evaporate, clear plates of cholesterin often crystallise out.

6. *Cystin* appears in the urine in the form of six-sided plates, which are insoluble in water and in acetic acid, but which readily dissolve in hydrochloric acid and ammonia. The pathology of cystinuria is very obscure.

### Sediments of Alkaline Urine.

The inorganic sediments which are found in alkaline urine may consist of various salts of phosphoric acid, of carbonate of lime, or of urate of ammonium.

1. *Amorphous Phosphate of Lime* forms a whitish flocculent deposit, which is not dissolved by heat, but at once passes into solution on the addition of a drop or two of acetic or nitric acid. Under the microscope this deposit is seen to consist of fine granules, arranged usually in irregular groups. In microscopic appearance they closely resemble amorphous urates, but the reaction of the urine will at once indicate their nature.

2. *Crystallised Phosphate of Lime (Stellar Phosphate)*.—The crystals of this salt are found in the urine in the form of rods, which either lie separately or are united to one another so as to form rosettes or sheaf-like bundles. This is a somewhat rare deposit, and is said to possess a graver significance than is attachable to the other varieties of phosphates.

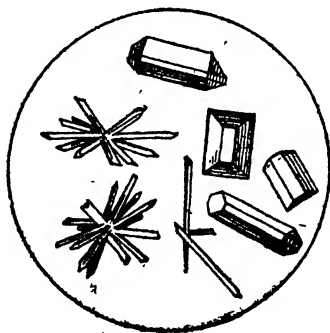


FIG. 43.—Triple Phosphate and Stellar Phosphate.

3. *Phosphate of Ammonium and Magnesium (Triple Phosphate)*.—This salt forms com-

paratively large clear crystals which may frequently be recognised by the naked eye as bright sparkling points adhering to the sides of the glass. Examined microscopically, they are found to be of varying form, usually, however, having the shape of a triangular prism with bevelled ends, and presenting from above the appearance of a glass knife-rest. Most usually the deposition of these crystals is simply due to the ammoniacal decomposition of the urine.

4. *Phosphate of Magnesium* is occasionally, though rarely, encountered in the urine in form of crystals—flat tables elongated in shape, clear and glassy.

5. *Carbonate of Lime*.—In human urine carbonate of lime only occurs in an amorphous form. It dissolves in acetic acid with effervescence. In the urine of the horse it forms spheres marked with radiating lines.

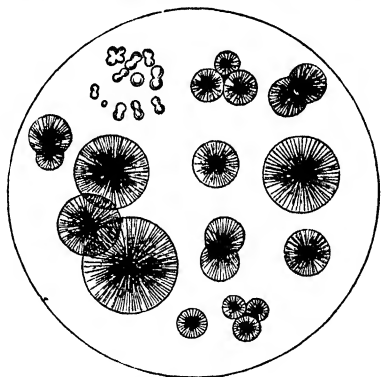


FIG. 44.—Carbonate of Lime (Roberts.)

6. *Urate of Ammonia* is found whenever the urine becomes strongly ammoniacal, in the form of opaque brownish spheres, which may be either smooth on the surface or may be covered with minute spikes. Sometimes this salt crystallises in the form of minute clear dumb-bells. It has no particular clinical significance.

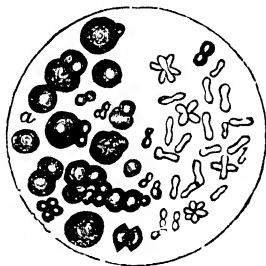


FIG. 45.—Urate of Ammonia (Roberts.)

## CHAPTER XXVIII.

### Nervous System.

#### SENSORY FUNCTIONS.

THE diseases of the nervous system are so complicated, and at the same time their diagnosis is now becoming in the majority of cases so precise, as a strict logical deduction from the signs and symptoms observed at the bedside, that very special attention must be paid to the systematic arrangement of the inquiries which the physician has to make, and to the methods to be employed in testing the condition of the various functions of the nervous apparatus.

These functions will be considered in the following order:—

1. Sensory Functions.
2. Motor Functions.
3. Vaso-motor Functions.
4. Trophic Functions.
5. Cerebral and Mental Functions.
6. Condition of Cranium and Spine.

#### SENSORY FUNCTIONS.

The phenomena met with in connection with the sensory apparatus are of two kinds; firstly, *subjective*—i.e., such sensations as arise independently of any external irritation; and, secondly, *objective*—i.e., sensibility to external stimulation of various kinds. Although this classification is open to consider-

able criticism, it will be found to be convenient for purposes of description, and for clinical examination.

**Subjective Sensations.**—Among the many sensations of which patients complain, the following are the most important:—

1. *Pain*.\*—Pain is simply an exaggeration of common sensibility. It is often the result of inflammation or other local disease of the tissues. When the pain is intermittent, darting, and occurs suddenly, following the course of some nerve and its branches, it is termed *neuralgia*. Of the many forms of neuralgia, the most commonly encountered are—

(a.) *Tic Douloureux*, neuralgia of the fifth nerve, which consists of paroxysms of pain corresponding in site to the nerve and its branches on the face.

(b.) *Intercostal Neuralgia* is, as its name implies an affection of the intercostal nerves. It is to be carefully distinguished from the pain of pleurisy and of muscular rheumatism.

(c.) *Sciatica*.—The pain here corresponds to the course of the sciatic nerve and its branches. It is usually localised between the tuber ischii and the great trochanter, and shoots downwards sometimes as far as the heel.

(d.) *Visceral Neuralgiæ*.—These pains may be referred to the region of the heart and aorta (angina pectoris), of the stomach, intestines, liver, kidneys, uterus, or ovaries.

It was first pointed out by Valleix that, when the nerve which is affected with neuralgia lies superficially, there can be found during an attack certain points upon the skin which are painful on pressure. Such painful points are usually situated where the nerve leaves an osseous canal or comes through a strong fascia.

\* Many of the varieties of pain mentioned are not subjective, arising as they do from local causes, but they are classified here because they occur without any stimulation of the sensory apparatus on the part of the physician.

Besides these neuralgiæ there are various painful sensations which are met with in nervous diseases, and which must be looked for in such cases. Of these we may notice—

(a.) *Girdle Pain*, which is the sensation of having a cord or girdle tied tightly round the body. It may be felt at various levels, on the thorax, abdomen, or on the lower extremities at the knee or ankle. It is commonly met with in connection with inflammatory and degenerative changes in the cord, particularly in cases of locomotor ataxia, and is believed to result from slight excitation of the posterior nerve-roots.

(b.) *Lightning Pains*.—Among the most common symptoms in locomotor ataxia, are paroxysms of darting, lancinating pains, which shoot through the body, and which have received the name of lightning pains.

(c.) *Headache* in all its many forms. Headache arises from a great variety of morbid conditions, such as vaso-motor changes within the cranium, abnormal composition of the blood, organic disease of the cranium or scalp, or of the brain or its membranes. The differential diagnosis of these different forms is to be found in special works on the subject. It is sufficient now to point out that the mode of invasion, the intensity, and the site of the headache must be exactly ascertained, as well as the presence of any obvious exciting cause.

2. *Paræsthesiæ*, or perverted sensations, are commonly met with among disorders of the nervous system.

(a.) *Sensations of Heat and Cold* (independently of actual elevation or depression of temperature as ascertained by the surface thermometer) are met with in intermittent fever, and also in various nervous diseases.

(b.) *Numbness*. — Any condition tending to depress the activity of the cutaneous sensibility may give rise to sensations of this kind, where the patient feels as if he were walking on a soft carpet, and has a tingling sensation up the limbs. It may also take the form of—

(c.) *Formication*, or the sensation of ants crawling over the

skin. These two forms of paræsthesia are caused by affections of the nerve trunks (cold, mechanical injuries, &c.), or of the central organs (locomotor ataxia, hysteria, &c.), and perhaps sometimes of the peripheral terminations. Formication likewise arises occasionally after the administration of morphia, aconite, and ergot.

(d.) *Pruritus*, or itching, is caused by disease or irritation of the terminal end organs in the papillæ of the skin. It arises as a result of—

(a.) Many skin diseases, particularly the parasitic varieties.

(b.) *Prurigo*.

(c.) Various chemical substances circulating in the blood—bile, sugar, hippuric acid, and perhaps xanthin and creatin.

3. *Giddiness (Vertigo)*, is a sensation of swimming in the head, the body appearing to oscillate in different directions, and surrounding objects to rotate, and is accompanied with reeling and staggering. In Ménière's disease, and in disease of the cerebellum, vertigo is a frequent symptom; but it also arises from any condition which disturbs the circulation in the cranium, such as dyspepsia, heart disease, mental work, &c. &c. In many cases vertigo arises from a contradiction between the impressions of external relations derived from two or more special senses.

4. *Abnormal Visceral Sensations*.—These comprise such sensory disturbances as pyrosis or water brash, boulimia or abnormal hunger, polydipsia or excessive thirst, and certain other similar symptoms. These have been already discussed in other parts of this work.

Such are the more important of the abnormal sensations complained of by patients suffering from nervous disorders. We now turn to what is of much greater value in diagnosis, in that it admits of precise determination—viz., the actual condition of the cutaneous sensibility as tested by the physician himself.

**Cutaneous sensibility is of two kinds—**

(1.) *Common or General Sensation*, which includes the consciousness of contact with a substance which, when exaggerated, amounts to pain ; and

(2.) *Tactile Sensation*, comprehending—

(a.) Sensations of pressure.

(b.) „ temperature.

(c.) „ locality.

These will each be considered in turn.

1. **Common Cutaneous Sensibility** may be tested by means of touching, tickling, pinching, or pricking the skin. All these methods of stimulation give indications, but by far the most accurate results, as Duchenne first showed, are obtained by using the Faradic current. The strength of the current may be varied from that just sufficient to cause a perceptible sensation up to one which produces sharp pain. By comparing the minimum strength of current required to cause a sensation at different parts of the body, important indications may be obtained.

2. **Tactile Sensibility** includes sensations of pressure, temperature, and locality.

(a.) *The Sense of Pressure* is most readily tested by the method devised by E. H. Weber, which consists in the application of different weights over the portion of skin to be examined. In order to eliminate the muscular sense it is necessary to lay the hand or foot, as the case may be, upon a firm support, and further, it is advisable to interpose some non-conducting substance, so as to prevent the temperature or size of the weight from being recognised, as these impressions might give some indication of its weight. Very rapid and accurate results can be obtained by the use of Eulenburg's *Baræsthesiometer*. This instrument is of simple construction, consisting essentially of a rod terminating in a vulcanite plate which is pressed upon the skin. The rod is so arranged that

when it is pressed up into the frame in which it is held it comprises a spiral spring and at the same time indicates on a dial the amount of tension.

(b.) *The Sense of Temperature* is tested by applying to the skin two bodies of unequal temperature, the most convenient being two test-tubes, one filled with cold, the other with hot water. The most sensitive parts of the body are the skin of the face and of the back of the hands (Weber), where differences of temperature of  $0.3^{\circ}$  C. can readily be distinguished. A difference equal to a degree of the centigrade scale can be appreciated at any part of the body.

(c.) *The Sense of Locality* is to be tested by pinching some portion of the patient's body when his eyes are closed, and making him indicate the site of the touch. In health the error is very small indeed. It may further be tested by ascertaining to what distance the two points of a pair of compasses, or of Sieveking's æsthesiometer, pressed upon the skin, must be separated before they can be recognised by the patient as distinct. Weber gives the following as the minimum distances to which the points must be separated, to be felt as different points, in health :—

On the point of the tongue, . . . . .	1.18 m.m.
„ palmar surface of last phalanx of finger, . . . . .	2.25 m.m.
„ „ „ 2nd „ „ . . . . .	4.5 m.m.
„ plantar „ last „ great toe, . . . . .	11.25 m.m.
„ back of the hand, . . . . .	31.5 m.m.
„ forearm and leg, and dorsum of foot, . . . . .	40.5 m.m.
„ upper arm and thigh, . . . . .	77.5 m.m.
(1 m.m. = 0.039 inch.)	

In disease all these various forms of cutaneous sensibility may be affected, either diminished (anæsthesia) or rendered more acute (hyperæsthesia), and they may be either collectively or individually affected.

When sensibility to pain is diminished, the condition is named *analgesia*, and when it is increased, *hyperalgesia*. In



the same way, we speak of *thermo-anæsthesia* and *thermo-hyperæsthesia*, when the sensibility to heat is diminished or increased; and other refinements of nomenclature have been devised by Eulenburg, such as *apselaphesia* and *hyperpselaphesia*, referring to tactile sensibility alone, the former indicating a decrease and the latter an increase. All these partial affections of the cutaneous sensibility are most frequently met with in cases of locomotor ataxia.

Finally, we have to note the rapidity of the conduction of these cutaneous sensory impressions to the brain. In health this takes place in a little over .1 second; and it is often easy—in the case of locomotor ataxia, for example—to observe an abnormal prolongation of this period. It is not uncommon to notice in such patients that the tactile and painful impressions do not travel at the same rate of speed, the prick of a needle being instantaneously felt as a touch, and, after an interval of a few seconds, as a painful sensation.

**Muscular Sense\***—that collection of impressions by means of which the mind is enabled to appreciate the degree of contraction of the muscles—demands careful investigation, since its presence or absence in a particular case is of great diagnostic significance. It is tested by causing the patient to lift various weights (suspended in a towel, so as to eliminate as far as may be the cutaneous sensibility to pressure), and we may either determine the smallest weight which can be appreciated as such, or the smallest difference between two weights which he can perceive. The muscular sense may also be tested by asking the patient, with closed eyes, to point to or to touch some particular part of the body, such as the tip of the nose or ear, or the great toe. The absence of this sense is frequently associated with loss of equilibrium of the body.

\* The common sensibility of the muscles is not here alluded to, as being of minor importance and possessing little diagnostic significance.

We now pass to the consideration of the special senses.

### SIGHT.

For a full description of the various affections of sight, special works on the subject must be consulted. A very brief outline may, however, here be given of the alterations which arise as the result of nervous affections, it being understood that all defects of vision caused by abnormalities in the refracting media are excluded from consideration.

*Optic Hyperæsthesia* and *Hyperalgesia*, the increase of the retinal sensibility — amounting to a painful sensation, with intolerance of light—is common in meningitis and in cerebral hyperæmia, and illusions of sight often occur in insanity, dependent upon central changes. The most frequent pathological condition, however, is

*Optic Anæsthesia*, the diminution or total loss of vision. When (from nervous causes) the sight is merely impaired, we speak of *amblyopia*; when it is entirely lost, of *amaurosis*.

In *amblyopia* the vision may be affected as regards acuteness, extent, or colour.

1. *Diminution in Visual Acuteness* causes the objects seen to appear blurred, misty, and indistinct. The most usual method of testing the acuteness of vision is by means of test types, which the patient is made to read at a distance. Defective vision may be merely functional, resulting from debility, gastric and uterine and renal affections, abuse of tobacco, &c., when no abnormal ophthalmoscopic appearances present themselves; or it may be due to optic neuritis or retinitis, or to atrophy of the optic nerve.

2. *Alterations in the extent of the Visual Field* are most accurately determined by means of the perimeter, but, for ordinary purposes, it is only necessary to stand opposite to and near the patient, who should be directed to shut one eye and to fix the other steadily on that of the operator. The latter should then move his hand in every direction, and ascertain at what

point it enters the visual field. Any deviation from the normal condition can be thus readily detected. The visual field may be encroached upon either from the centre or from the margin. In the former case a dark spot (scotoma) forms in the centre and gradually enlarges. The scotoma may be steady or scintillating, zig-zag, and brightly coloured, as in migraine. Such limitations of the field of vision are met with in functional amblyopia (especially from abuse of tobacco), in optic neuritis and in optic atrophy.

The field of vision may further be abolished as regards one lateral half of each of the retinae (hemiopia), the vertical line of demarcation being very sharply defined. This affection of vision occurs in cases of disease of the optic tracts, of the commissure, of the corpora geniculata, the thalamus, and the cortex of the occipital lobes. The exact localisation of the lesion can be deduced from the anatomical arrangement of the nerve fibres in the tracts and commissures, but for details special works must be consulted.\*

3. *Alterations in the Perception of Colours.* — To test the perception of colours, a scale of colours as those of the spectrum should be employed, and the patient should be called upon to name them when presented to him. Colour-blind persons cannot distinguish red. The visual field for the perception of colours may also be encroached upon. In health it is to be noted that the area of vision differs in the case of each colour, being greatest for white—next yellow, then blue, red, and, last of all, green.

#### MOVEMENTS OF THE EYEBALL.†

The ocular muscles are supplied by three nerves—the oculomotorius, or 3rd nerve; the trochlearis, or 4th nerve; and

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\* In particular, Charcot's "*Leçons sur la Localisation des Maladies du Cerveau.*"

† Strictly speaking, these ought to be considered among the motor functions, but for convenience they are introduced here.

the abducens, or 6th nerve. Spasm and paralysis of each of these affect the muscles which they supply, and produce corresponding changes in the position of the eyeball. Their action on the pupil will be subsequently considered.

### I.—Paralysis of the Ocular Nerves.

(1.) *Oculo-motorius, or 3rd Nerve*.—According as the paralysis is complete or incomplete, the whole, or only one or more of the following muscles are affected:—

(a.) *Levator Palpebræ Superioris*.—Paralysis causes drooping of the upper eye-lid—ptosis.

(b.) *Superior Rectus*.—The eyeball turns downwards and slightly outwards when this nerve is paralysed, and there is in consequence diplopia or double vision, the result of the visual axis of the two eyes not being directed to the same object. As this divergence does not exist when the eyeballs are both turned downwards, the patient instinctively carries the head well thrown back.

(c.) *Internal Rectus*.—Paralysis here gives rise to divergent strabismus (squint), with diplopia, the eyeball being rotated outwards on account of the unopposed action of the external rectus.

(d.) *Inferior Rectus*.—The affected eye is, in paralysis of this muscle, directed upwards and slightly outwards, and there is diplopia except when the object lies above the level of the eyes.

(e.) *Inferior Oblique*.—In paralysis of this muscle the eyeball is turned slightly downwards and inwards, but this condition is rarely observed, as paralysis of the inferior oblique as an isolated affection is exceedingly uncommon.

(f.) *Ciliary Muscle*.—The effect of paralysis of this muscle is that the patient is unable to change the convexity of the anterior surface of the lens, or, in other words, to focus his eye for near objects.

When the 3rd nerve is paralysed as a whole all these actions combine, and the result is, that the lid droops, the eyeball is drawn downwards and outwards, and protrudes from its socket, the pupil (as we shall presently see) is dilated and immobile, and there is defective power of accommodation.

(2.) *Nervous Trochlearis, or 4th Nerve*, supplies the superior oblique muscle, and when that is paralysed there is diplopia, the field of vision being moved downwards and outwards.

(3.) *Nervous Abducens, or 6th Nerve*, supplies the external rectus, paralysis of which causes convergent strabismus, with consequent diplopia, there being no power of rotating the eyeball outwards beyond the middle line. This condition frequently gives rise to giddiness, nausea, and vomiting.

Paralysis of the ocular nerves may be either central or peripheral. In the latter case the paralysis is usually more complete. Ocular paralysis is common in locomotor ataxia, syphilis, hemiplegia, &c., and after diphtheria.

## II.—Spasm of the Ocular Nerve.\*

### (1.) *The Third Nerve*—

(a.) *Levator Palpebræ Superioris*.—Tonic spasm of this muscle causes the eyelid to be drawn up, and the eye cannot therefore be closed. It results either from peripheral irritation (rheumatism, wound, &c.), or, reflexly, from irritation of the sensory fibres of the fifth nerve, or is due to central causes. Clonic spasm hardly ever occurs in this muscle.

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\* Muscular spasm (which will be hereafter described) is of two kinds—(1) *Tonic*, when the contraction remains of nearly uniform intensity while the spasm lasts; and (2) *Clonic*, when there is a condition of rapidly alternating contraction and relaxation.

(b.) *Internal Rectus*.—Of all the muscles of the eyeball this is the most frequently affected with tonic spasm. There is then convergent strabismus with diplopia, in which the two images are not at a fixed distance from each other (as in squint from paralysis of the 6th nerve), but undergo a constant oscillation, alternately approaching and receding from one another.

(2.) *The Sixth Nerve*.—Tonic spasm of the external rectus is rarely met with without implication of other muscles. When present the eyeball is drawn outwards, giving rise to divergent strabismus.

*Clonic Spasm* of the muscles of the eyeball generally gives rise to a condition named *Nystagmus*, when the eyeball undergoes continuous oscillatory and rotatory movements which cannot be controlled by the will. It arises either from local abnormalities of the ocular structures, or from central nervous affections, such as locomotor ataxia, insular sclerosis, meningitis, hydrocephalus, &c. Coal-miners are frequently affected with nystagmus, owing to the constrained position and bad light in which they have to work. In them the nystagmus is probably strictly analogous to writer's cramp in clerks, and to trade spasms generally.

#### CHANGES IN THE PUPIL.

The iris being under the control of two mechanisms, the one reflex (contracting)—the afferent fibres lying in the optic nerve, the efferent in the 3rd nerve, and the centre in the corpora quadrigemina; and the other a dilating mechanism—the efferent fibres being in the sympathetic and the centre in the cilio-spinal region of the cord (lower cervical and upper dorsal); so spasm or paralysis of any of these structures will give rise to changes in the size of the pupils. There is probably, however, a third mechanism—viz., a local centre for the movements of the iris, lying either in the iris itself or in the neighbouring choroid, and on this mechanism act various drugs when dropped

on the conjunctiva, such as atropin (dilating) and physostigmin (contracting).

The most common causes of change in the pupils are as follows :—

*Dilatation of the Pupils* is met with—(1.) in cases of coma (except from opium) ; (2.) in cases of glaucoma and of myopia ; (3.) from paralysis of the third nerve ; (4.) from irritation of the sympathetic nerve, as from aneurism ; (5.) from the action of atropine, local or general ; (6.) from lesions of the optic nerve ; (7.) in children, and nervous and chlorotic subjects.

*Contraction of the Pupil* is common in old persons. It further occurs in connection with—(1.) paralysis of the sympathetic, as from pressure of aneurism ; (2.) disease of the cilio-spinal region in locomotor ataxia ; (3.) retinitis, iritis, &c. ; (4.) the action of morphia, tobacco, and physostigmin ; (5.) hypermetropia ; (6.) increased intracranial pressure, as from tumours ; (7.) lesions of the pons and medulla oblongata.

*Hippus*, or clonic spasm of the iris, shows itself in quickly alternating contraction and dilatation of the pupil. It is sometimes seen in cases of paralysis of the 3rd nerve, and probably in disease of the sympathetic.

*Argyle-Robertson Symptom* consists in the absence in the pupil of any response when the retina is stimulated by light, while at the same time it moves normally when accommodation is made. This condition is frequently met with in locomotor ataxia.

#### OPHTHALMOSCOPIC EXAMINATION.

The use of the ophthalmoscope ought to be a matter of routine in all cases of nervous disease, for the condition of the optic nerve and retina often throws much light on an otherwise obscure diagnosis. Of the many forms of apparatus which have been devised, probably the most convenient is that of Dr. Gowers. It consists essentially of a concave mirror to throw the rays of light into the interior of the eye to be examined, pierced in the centre with a small hole through

which the observer looks. Behind this mirror is fixed a disc bearing a series of convex and concave lenses, which can in turn be brought so as to lie over the aperture in the centre of the mirror, and thus come between the eye of the physician and that of his patient. There are also supplied along with the instrument two larger, bi-convex, lenses which are to be used as will be presently described. The ophthalmoscopic examination of the retina may be conducted in two ways, either with the simple mirror—giving an upright image,—the direct method, or with the mirror along with a lens—giving an inverted image,—the indirect method, as in fig. 46.

1. *Upright Image.*—The patient is placed in a dark room with a bright light—either gas or oil—at the side of his head on a level with the eye. The physician now takes up the ophthalmoscopic mirror, and resting its upper edge upon his eyebrow and looking through the central aperture, he inclines it so as to throw a strong beam of light into the eye of the patient. If the refraction of the eyes, both of the patient and of the observer, be normal, nothing more is required than that the latter should bring his eye close to the patient and relax his accommodation, looking as it were at a distant object. The retina will then come into view as an upright image. Should the patient's eye be hypermetropic, it may become necessary, in order to obtain a distinct image, to employ a convex lens to counteract the divergence of the rays as they emerge from the hypermetropic eye. Suitable convex lenses are fixed in the rotating disc behind the mirror, and this disc should be turned, bringing lens after lens between the two eyes, until the one which suits the particular case has been found. In the same way in cases of myopia a concave lens must be interposed.

2. *The Inverted Image.*—To use the ophthalmoscope to obtain an inverted image of the retina, the observer places himself at a little distance from the patient, say about a foot off, and holding the mirror as before, he throws the light upon the eye to be examined. In his unemployed hand he holds a



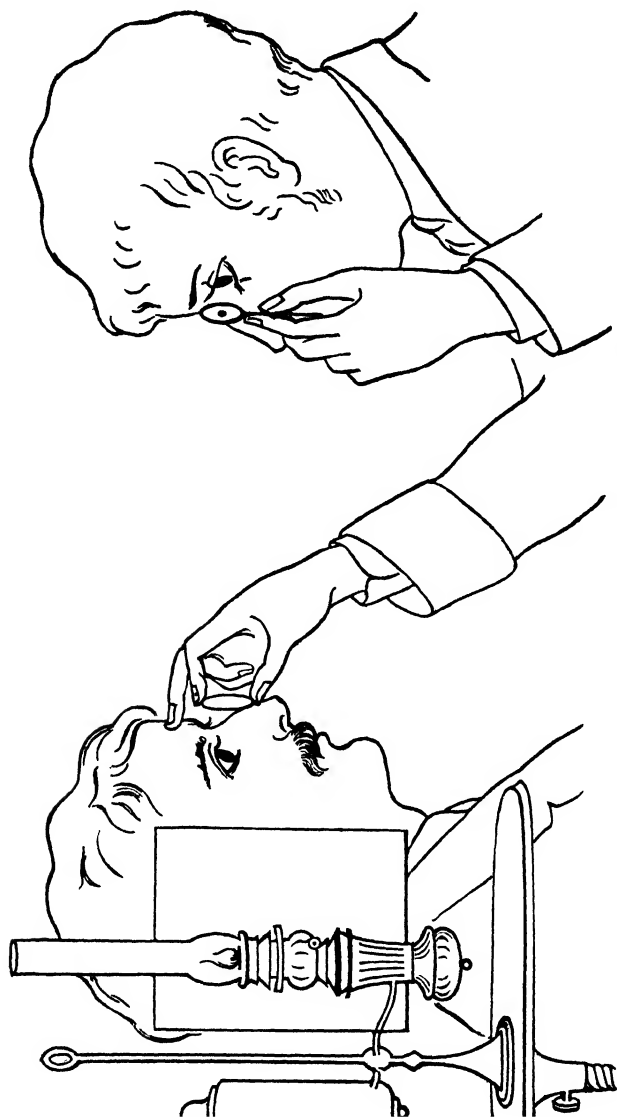


FIG. 46.—Indirect method of using the Ophthalmoscope.

bi-convex lens between the thumb and index finger, and places it vertically between his eye and that of the patient, at a short distance, about  $1\frac{3}{4}$ -2 inches, from the latter. The lens will be held with greater steadiness if the small finger be allowed to rest on the eyebrow of the patient. In this way an inverted image of the retina is obtained.

Both these methods of using the ophthalmoscope should be made use of, as each has its special advantages. With the first, the area of the retina seen at one time is circumscribed, but is considerably magnified; with the second, the magnifying power is small, but a great deal more of the retina comes into view at one time. The use of atropine or homatropine to dilate the pupil ought to be, as far as possible, avoided. The instances are few in which the information required for diagnosis of diseases, other than those of the eye itself, cannot be obtained without preliminary dilatation.

*Ophthalmoscopic appearance of the normal Fundus of the Eye.*

The normal appearance of the fundus varies so much in different people, that it requires very considerable practice to be able to say that any individual eye is natural. It is best to make the examination methodically, commencing by inspecting the optic disc or papilla, and then passing to the retina with its blood-vessels, and to the choroid.

*The Optic Papilla* is usually elliptical in shape, the result of the angle at which the optic nerve enters the eyeball. The limiting margin of the papilla is formed by the sclerotic and choroid through which the nerve pierces its way to gain the interior of the eye, and inasmuch as most usually the aperture in the choroid is somewhat larger than that in the sclerotic, the latter membrane frequently shows as a faint bluish ring round the papilla—the sclerotic ring. At the edge of the choroid there is very often a deposit of pigment, more or less extensive (shown to a slight degree in fig. 48), which must not be mistaken for a pathological condition. The

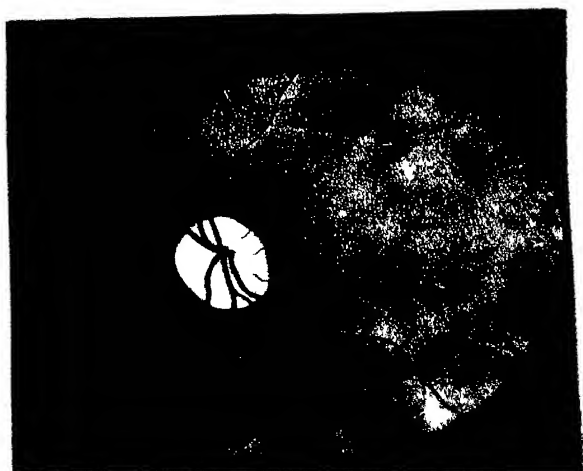
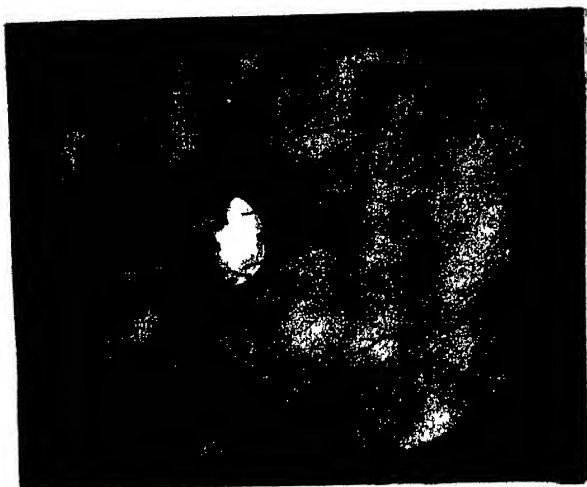
FIG. 47.—THE NORMAL FUNDUS.

Both arteries and veins stand out a little too distinctly in the photograph, and the latter appear rather broader than they should do.

FIG. 48.—ATROPHY OF THE OPTIC NERVE.

The papilla is of a dull white, and its edge at some points not well defined. The central vessels are distinctly diminished in size.\*

\* For the opportunity of making the drawing from which this photograph was taken, I am indebted to the courtesy of Dr. Argyle Robertson, from one of whose patients the sketch was made.





*colour* of the papilla is very difficult to describe. It is for the most part white, but derives a very faint greenish lustre from the nerve fibres, and a somewhat more distinct pink from its blood-vessels. On the whole it may be said to be faintly pink. The outer half of the papilla is more white than the inner, owing to the fact that the nerve fibres are chiefly distributed on the inner side, and as a consequence the blood supply is there more abundant. The central area of the papilla is more pale than any other part, and is almost always slightly excavated—the *physiological excavation*—on account of the divergence of the nerve fibres which there takes place. The excavation differs very much as to depth in different people, often showing at its lowest part the faint blue colour of the *lamina cribrosa*. Out of this excavation comes the central artery of the retina, and into it disappears the central vein—the branching of both of these vessels varying somewhat in different cases, but almost invariably one branch passes upwards and the other downwards.

*The Retina* is only visible to the ophthalmoscope by reason of its blood-vessels. It is perfectly transparent, except in some persons in the region of the macula. The retinal vessels follow a fairly uniform course, always being more abundant on the inner side where most nerve fibres lie. Each artery is accompanied by one vein, which is deeper in tint and about one-third broader than the artery. The walls of the blood-vessels are invisible in the healthy fundus, it being only the column of blood which shows, but from the peculiar way the light is reflected from each red column, the edges appear as dark lines. The nerve fibres follow the same course as the arteries, but are almost always invisible, as they lose their sheath of myelin just as they enter the papilla. Occasionally, however, individuals are met with in whom some of the nerve fibres have retained their sheaths, and thus appear as brilliant white lines, stretching in patches, very much in the same line as the blood-vessels. In some persons the macula is invisible; in many, however, it appears dark-red, and surrounded with one or more oval white rings.

*The Choroid.*—The general tint of the fundus is chiefly due to the vessels and pigment of the choroid, the pigment giving rise to the more or less dark appearance of the ophthalmoscopic image. The amount of choroidal pigmentation varies much in different individuals, in dark-complexioned and dark-haired persons being deep, and in blondes showing little. In the latter case the pigment is so slightly deposited as to allow the blood-vessels of the choroid to appear, and this is still more marked in albinos.

#### *Diseases of the Optic Nerves.*

The diseases of the optic nerves which can be recognised by means of the ophthalmoscope are mainly these—(1.) Congestion, (2.) Optic neuritis, (3.) Optic atrophy.

1. *Congestion of the Papilla* shows itself by an increased redness of the disc. The margin of the papilla loses its sharpness, and becomes more or less blurred and indistinct.

2. *Optic Neuritis.*—In this condition the red colour of the papilla deepens, and its edge becomes very indistinct, so much so as often to be unrecognisable. Along with these changes there is associated an cedematous swelling of the papilla (the so-called choked disc), the veins are much engorged and tortuous; the arteries, on the other hand, are distinctly reduced in size. In well-marked cases the swollen papilla forms a prominent tumour with steep edges over which the blood-vessels disappear from view, to reappear in a different line on the neighbouring retina. Hæmorrhages frequently take place into the swollen papilla. The most common cause of optic neuritis is undoubtedly cerebral tumour, but the condition is occasionally found in connection with hydrocephalus, rarely in cerebral abscess and cerebral embolism. Optic neuritis may also be occasioned by various pathological conditions within the orbit, and by such acute diseases as typhus, pneumonia, or scarlet fever. There is a special form of optic neuritis met with in cases of Bright's disease, which will be mentioned in connection with albuminuric retinitis.

3. *Atrophy of the Optic Nerve.*—The variety of the optic atrophy which is called simple or primary, that met with in connection with lesions of the posterior columns of the cord and of the brain, is characterised by a peculiar whitish or greenish colour in the disc, due partly to diminution of the capillary circulation and partly to the disappearance of nerve fibres and the increase of connective tissue. The papilla is sharply defined and flat; but the central artery and vein are not appreciably affected. When the atrophy is the result of preceding neuritis, the disc is white or faintly yellow, and is more or less obscured in outline by exudation. The central vessels are often varicose, and are diminished in size. If the atrophy be the result of embolism or thrombosis of the central artery the disc appears pearly white, slightly obscured at the margin, and the central artery and its branches are so much diminished in size as only to appear as fine lines. Atrophy of the disc is also met with in connection with choroiditis and retinitis pigmentosa, and as a result of glaucoma. In the last-named affection the increase of intra-ocular pressure presses back the optic nerve in such a way as to give rise to a deep excavation in the papilla, and to cause atrophy of the nerve tissue.

#### *Diseases of the Retina.*

1. *Papillo Retinitis.*—When the lesion of the papilla which has just been described as optic neuritis advances to a certain point of intensity, the retina itself becomes affected, being infiltrated with transudation. The veins are much engorged, and small hæmorrhages result. These lie, as a rule, in the neighbourhood of the disc, and though of no great size can be readily recognised. The retinal nerve fibres degenerate and become visible at scattered points of the fundus as opaque white patches, and the perivascular tissue undergoes proliferation, and hence the vessels become bounded with white lines.

2. *Albuminuric Retinitis.*—In cases of Bright's disease there is often found a degree of retinitis resembling very closely the



papillo-retinitis already described. The cases are, however, peculiar in respect of the amount of hæmorrhage and the rapidity with which the extravasated blood undergoes fatty degeneration. There are thus left numerous white patches which are often arranged in a peculiar and characteristic stellate manner. A similar form of retinitis sometimes occurs in connection with diabetes.

3. *Pigmentary Degeneration of the Retina.*—In this very peculiar condition the surface of the retina is scattered over with small masses of black pigment, arranged chiefly round the blood-vessels. There is well-marked atrophy of the optic disc, and great diminution in the calibre of the central vessels.\*

## HEARING.

In connection with this sense we often meet with subjective sensations, which from their persistence are frequently complained of by the patient. These consist of roaring, humming or ringing sounds (*tinnitus aurium*) which may or may not be accompanied with giddiness. In the majority of cases such symptoms result from disease of the middle ear, and here fall within the domain of the surgeon. When such is not the case, they may arise from affection of the inner ear, of the auditory nerve, or of the central organs in the brain. The first of these is met with in cases of Ménière's disease, in which ringing in the ear is accompanied with loss of equilibration. Although the nerve fibres engaged in transmission of sound impressions and those in connection with the sense of equilibrium run for some distance together, yet it sometimes occurs (as in locomotor ataxia) that the latter are alone affected, so that auditory vertigo results without any deafness. In the brain these two sets of nerves divide to proceed to different centres, and thus, when

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\* For a full description of the various ophthalmoscopic appearances met with in the course of medical diagnosis, the reader must be referred to special works, such as "Gowers' Medical Ophthalmoscopy," or the "Ophthalmoscopie Clinique" of De Wecker and Masselon.

subjective sensations of hearing result from central brain causes, we find that where the auditory centre is affected we have tinnitus or deafness and no giddiness, whereas when the affection is in the cerebellum the conditions are reversed, giddiness and not deafness resulting. Ringing in the ears is, as is well known, frequently observed to result from the administration of quinine and salicylic acid; and, finally, it must not be forgotten that tinnitus is common where there is anæmia, being then perhaps not purely subjective but probably resulting from the anæmic murmurs in the vessels to which reference has been made in a former chapter.

Passing now to the physical examination of the ear, it may be briefly said, that so far as affections of the outer and middle ear are concerned, the method of examination should be conducted by means of a speculum, light being reflected into the instrument from a concave mirror held by the examiner. For details of manipulation special works on the subject should be consulted;\* in which, also, information regarding changes in the appearance of the tympanic membrane will be found. The condition of the Eustachian tube should also be ascertained.

*Perception of Sound Waves.*—The degree of acuteness with which sound waves are perceived in an individual case is ascertained by means of a watch. The normal distance at which the ticking of the particular watch employed can be heard, should first be noted. The watch is then held at that distance, and gradually brought nearer to the patient's ear until the sound is perceived. Having ascertained this distance, it is then advisable to test the patient's power of perceiving a whispered voice—each ear being tested separately. For medical purposes, however, the third test is the most important. It is directed to ascertain the condition of the nervous apparatus in the inner ear, and is performed by means of a tuning-fork. If the stem of a vibrating fork be applied to the vertex or to

\* See Dr. P. M'Bride's "Guide to the Study of Ear Disease."

the teeth, the vibrations are communicated through the bones directly to the labyrinth, and in normal conditions are perceived equally on both sides. If this is so, we may safely conclude that the terminal nerve organs of hearing are intact, and this even if deafness to ordinary sound exist. In such a case the deafness must be due to some affection of the middle or external ear, and the very obstruction which prevents sound from reaching the labyrinth equally prevents sound from escaping, so that when the fork is applied to the head, the sound is heard most loudly in the deaf ear; unfortunately, however, the converse is not invariably true. In persons below the age of forty we may indeed conclude that if the tuning-fork is not well heard on the vertex, the auditory nervous apparatus is at fault, but after that age a degree of bluntness in the perception of these vibrations is not uncommon.

Hyperæsthesia of the auditory nerves is common in hysteria, in acute febrile diseases, and in insanity. It may also result from paralysis of the stapedius muscle with consequent over-tension of the *membrana tympani*, in cases of facial paralysis where the lesion lies above the origin of the branch to that muscle. More important clinically is the subject of auditory anæsthesia. In the great majority of cases deafness depends upon disease of the outer or the middle ear. We have already shown how nervous deafness is to be distinguished from these. The diagnosis of affections of the auditory nerve in its course, and of its centres can only be made by means of the other symptoms.

#### TASTE.

The sense of taste is located in the surface of the tongue, fauces, and back wall of the pharynx. The root of the tongue (circumvallate papillæ), fauces, and pharynx are supplied by the glossopharyngeal nerve. The taste nerve for the anterior two-thirds of the tongue, on the other hand, is the lingual, and the majority, if not the whole of the sensory fibres of taste, pass from the lingual into the chorda tympani, and then to the

facial, which nerve, however, they leave below the geniculate ganglion, to join (by channels at present unknown) the fifth nerve, in the trunk of which they pass to the brain.

To test the sense of taste the patient should be made with closed eyes to protrude his tongue, on different points of which the substances in solution used in testing are to be deposited by means of a glass rod. For *bitter* tastes, solutions of quinine, picric acid, and infusion of quassia may be employed; for *sweet*, syrup is the most convenient; *acid* taste will be produced by the application of vinegar or dilute acids; and *saline* by means of solutions of common salt, or of bromide or iodide of potassium. Sweet tastes are best felt at the tip of the tongue, acid at the edges, and bitter at the root of the organ. Perhaps, however, the most accurate method of testing the sense of taste is by means of a galvanic current. Hyperæsthesia of the sense of taste is rarely met with, but it occurs occasionally in cases of hysteria. Paræsthesiæ or abnormal sensations of taste are sometimes met with in insanity.

*Anæsthesia* of taste may be peripheral, due to a coating of fur on the tongue, or abnormal dryness of the mouth, or to the action of heat or cold. It may also be due to defective conduction, from disease of the nerves of taste in their course. In this way it may arise from lesion of the glossopharyngeal, when the defect of taste will be limited to the root of the tongue and fauces. When the anæsthesia involves the anterior two-thirds of the tongue, it is due to the fibres of the lingual nerve, the course of which has been already pointed out. The affection of these fibres has considerable diagnostic value. 1. If taste is thus lost along with loss of ordinary tactile sensation in the tongue, and without other indications of affection of the fifth nerve, then the lesion is in the lingual. 2. When the chorda tympani is alone affected, taste is lost on the anterior two-thirds of the tongue without tactile sensation being affected. This often occurs in connection with diseases of the middle ear. 3. Where facial paralysis accompanies the loss of taste, then the lesion is situate on the nerve between the geniculate

ganglion and the point at which the chorda tympani leaves the nerve. 4. When symptoms of affection of the second division of the fifth nerve accompany the loss of taste, the lesion lies on the nerve tract, between the spheno-palatine ganglion and the gasserian ganglion. 5. When the loss of taste is accompanied by total anæsthesia over the region supplied by the fifth nerve, the lesion lies at the root of that nerve on the base of the skull.

### SMELL.

The sense of smell is conveyed to the brain solely by the olfactory nerves. The branches of the fifth nerve distributed to the nasal mucous membrane have only to do with common and tactile sensation. To test the sense the patient may be made to smell various odoriferous substances, such as the essential oils, musk, camphor, valerian, &c.,\* or to hold in the mouth such articles as cheese, wine, and liqueurs, which owe their agreeable flavour to the sense of smell (the latter test is particularly useful when the nostrils have become occluded). Hyperæsthesia of the sense of taste is occasionally seen in hysteria. Loss of smell may be due to any cause which prevents the access of the aromatic particles to the mucous membrane, such as polypus, catarrh, abnormal dryness of the membrane (in paralysis of the fifth), or paralysis of the muscles necessary to the act of "sniffing" from paralysis of the seventh nerve. But apart from these causes, there is a true anæsthesia of the olfactory nerve (anosmia), which occurs in hysteria, tumour of the brain, embolism of the middle cerebral artery, blows on the head, and, as Althaus has pointed out, in locomotor ataxia.

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\* Such substances as ammonia and acetic acid should be avoided, as they only irritate the branches of the fifth nerve, and not the olfactory.

## CHAPTER XXIX.

### **Herbous System—(continued).**

#### **MOTOR FUNCTIONS.**

For practical purposes, the various motor functions may be arranged in the following manner:—

##### *A. Visceral Motor Functions.*

##### *B. Functions of Voluntary Muscles.*

##### **I.—VOLUNTARY MOVEMENTS.**

(1.) Paralysis.

(2.) Spasm.

##### **II.—REFLEX ACTIONS.**

(1.) Superficial Reflexes.

(2.) Deep Reflexes.

##### **III.—CO-ORDINATION.**

##### *C. Vaso-Motor Functions.*

##### **A.—Visceral Motor Functions.**

The movements of the viscera are regulated by means of so complex a nervous mechanism, and enter as yet so little within the scope of diagnosis, that they need only be very briefly alluded to here. There are, however, certain reflex actions which are of diagnostic value as indicative of the condition of that part of the spinal cord where their centres are situated.

(1.) *Deglutition* has already been spoken of under the heading

of the "Alimentary system." The reflex contractions of the œsophagus are in the main under the control of a centre in the medulla, and cease when that centre is diseased—as, for example, in advanced bulbar-paralysis.

(2.) *Micturition and Defœcation.*—The centres for these acts lie in the lumbar enlargement of the cord, and their performance is under the influence of the will. If from disease of the cord above the centres, volition is cut off, then, when the fœces have sufficiently accumulated, or the urine collected in the bladder to a certain amount, these excretions are expelled by means of reflex contraction. Involvement of the sensory tracts in the disease prevents the patient from being conscious of these acts. When the lumbar centres are themselves affected, then fœces and urine are evacuated so soon as they enter the rectum or bladder.

(3.) *Sexual Functions.*—These functions are controlled by a reflex centre in the lumbar enlargement of the cord, so close to that for the cremasteric reflex that the condition of that reflex affords us a trustworthy indication of that of the sexual functions. If the control of the higher centres is cut off by disease in the upper part of the cord, the process becomes imperfect, and may sometimes be excessive (priapism). Disease of the sexual centre in the lumbar enlargement causes loss of sexual power.

(4.) *Respiration.*—The respiratory centre lies in the medulla close to and below the vaso-motor, the *nœud vital* of Fluerens. In advanced bulbar-paralysis, it is sometimes attacked with disease, with a necessarily fatal result.

### B.—Motor Functions of Voluntary Muscles.

In examining the condition of the muscular system the following points should be investigated:—

1. Nutrition of the Muscles.
2. Tonicity of the Muscles.
3. Voluntary Muscular Movement.

4. Abnormal Muscular Movements.
5. Mechanical Irritability.
6. Reflex Irritability.
7. Electrical Irritability—Electro-diagnosis.

### 1. *Nutrition of the Muscles.*

This subject will be considered subsequently under the head of the Trophic Functions.

### 2. *Tonicity of the Muscles.*

The tension of the muscles is readily appreciated by examination with the hand—a rigid muscle being hard, a flaccid one soft to the touch. When the muscles of a limb are abnormally tense, there is a degree of resistance to passive movement which is readily appreciated, and affords an important indication. The intimate connection between the tonicity of the muscles and the tendon-reflex causes alterations in the latter when rigidity exists. This subject will, however, be again referred to.

*Flaccidity* of the muscles occurs where there is muscular atrophy. It is best marked, perhaps, in spinal cases, where the anterior cornua of the cord are involved in the diseased process.

*Rigidity* of the muscles, when it assumes a well pronounced form, is termed *contracture*. Of this condition there are two forms—an active and a passive. Active contracture is found as a result of increased reflex tonus or of direct irritation of motor nerve fibres. It occurs in cases of sclerosis of the pyramidal tracts, in meningitis, and in hysteria. The second form of contracture—the passive—is found where, from some joint injury for example, a limb has been long maintained in a bent position, so that the points of origin and insertion of the muscle in question have been kept nearer each other than in health. It also occurs when inflammatory changes or degenerative atrophy take place in the muscle. These two forms can usually be distinguished clinically. In active contracture the limb can usually be straightened without pain, and when left alone it



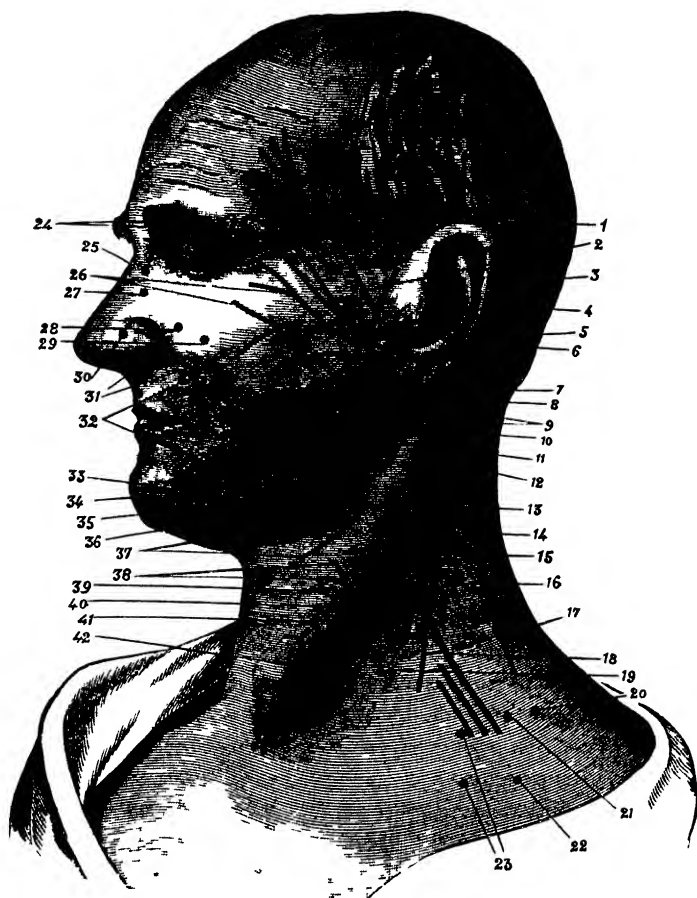


FIG 49 —Motor Points of Head and Neck. (Ziemssen )

## EXPLANATION OF FIG. 49.

1. Frontalis muscles.
2. Attrahens and attollens auriculam muscles.
3. Retrahens and attollens auriculam muscles.
4. Occipitalis muscle.
5. Facial nerve.
6. Posterior auricular branch of facial nerve.
7. Stylohyoid muscle.
8. Diaphragm muscle.
9. Buccal branch of facial nerve.
10. Splenius capitis muscle.
11. Subcutaneous branches of inferior maxillary nerve.
12. External branch of spinal accessory nerve.
13. Sterno-mastoid muscle.
14. Cucullaris muscle.
15. Sterno-mastoid muscle.
16. Levator anguli scapulæ muscle.
17. Posterior thoracic nerve (Rhomboidi muscles).
18. Phrenic nerve.
19. Omohyoid muscle.
20. Lateral thoracic nerve (Serratus magnus).
21. Axillary nerve.
22. Branch of brachial plexus (musculo-cutaneous and part of median).
23. Anterior thoracic nerve (Pectoral muscles).
24. Corrugator supercilii muscles.
25. Compressor nasi and pyramidalis nasi muscles.
26. Orbicularis palpebrarum muscle.
27. Levator labii superioris alæque nasi muscle.
28. Levator labii superioris muscle.
29. Zygomaticus minor muscle.
30. Dilator naris.
31. Zygomaticus major.\*
32. Orbicularis oris.
33. Branch to triangularis and levator menti muscles.
34. Levator menti muscle.
35. Quadratus menti muscle.
36. Triangularis menti muscles.
37. Cervical branch of facial nerve.
38. Branch to platysma muscle.
39. Sterno-hyoid muscle.
40. Omohyoid muscle.
41. Sternohyoid muscle.
42. Sternohyoid muscle.

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\* The upper of the two lines that converge on 31 should have been directed to 30 as it applies to the Dilator naris posterior muscle.

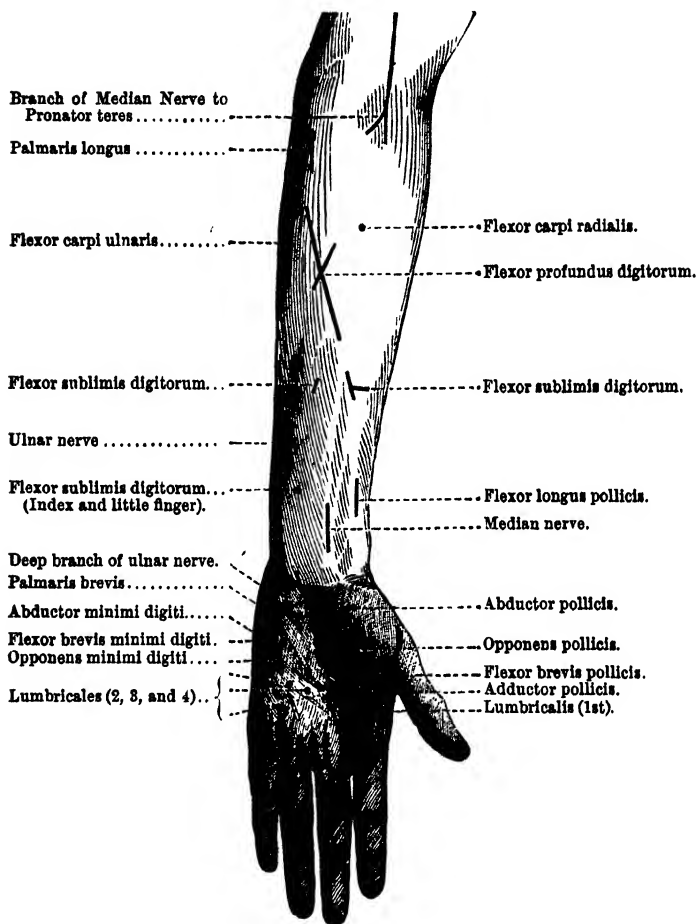


FIG. 50.—Motor Points of Forearm. (Ziemssen.)

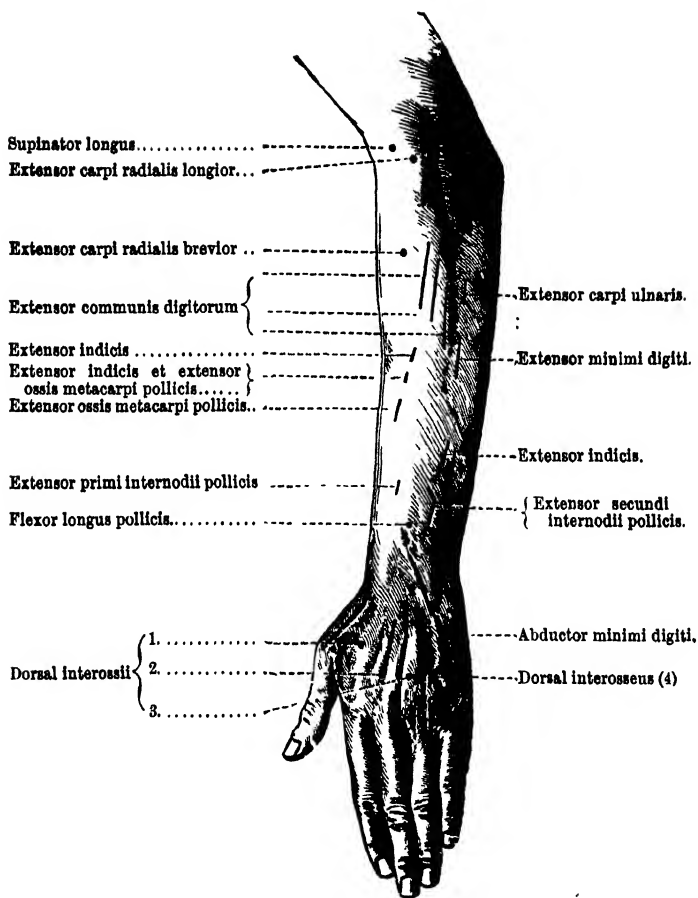


FIG. 51.—Motor Points of the Forearm. (Ziemssen.)

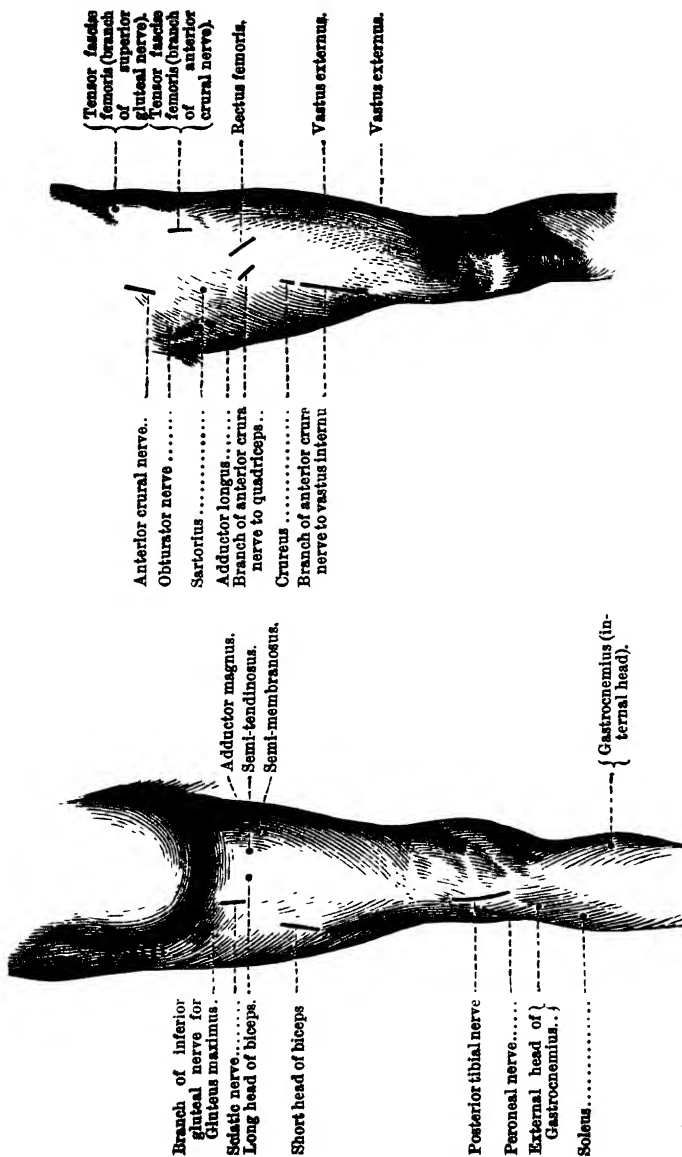


FIG. 52.—Motor Points of Lower Limb. (Ziemssen.)

FIG. 53.—Motor Points of Inferior Limb. (Ziemssen.)

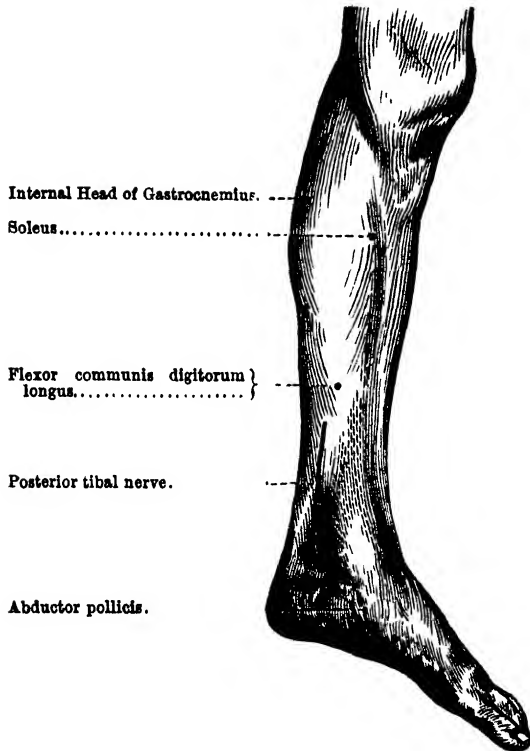


FIG. 54.—Motor Points of Inferior Limb. (Ziemssen )

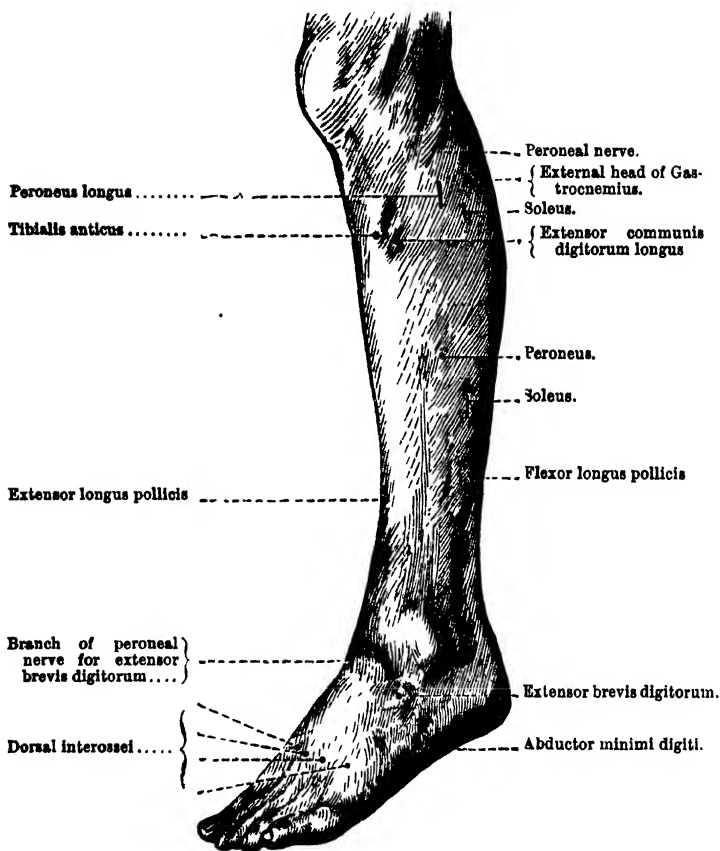


FIG. 55.—Motor Points of Lower Limb. (Ziemssen)

springs back quickly. It is increased by stimulation of the skin, relaxes during sleep, and disappears when chloroform narcosis is induced. In cases of passive contracture, on the other hand, the limb can hardly be straightened, and attempts to do so are attended with pain. When left to itself the limb very slowly recovers its position. The conditions of sleep and narcosis are without effect upon the degree of contraction.

### 3. Voluntary Movements of the Muscles.

When a muscle or group of muscles fails to respond, by shortening in the normal way, to a normal stimulus—we speak of *paralysis* (akinesis)—the lesser grade of which is called *paresis* (hypokinesis).

*Paralysis* varies much in regard to its extent and distribution. It may be limited to one or two muscles, or it may affect all the muscles of one lateral half of the body (hemiplegia), or of both sides of the body symmetrically, usually both lower limbs (paraplegia). When the paralysis is limited to one group of muscles or to one limb, we speak of monoplegia.

To examine the state of the muscular system, it is necessary to cause the patient to go through all varieties of voluntary

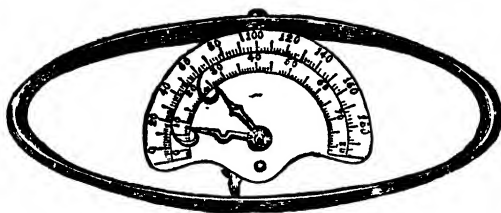


FIG. 56.—Dynamometer.

movement, simple and combined, standing walking, stepping up upon a chair, &c., as well as such actions as speaking,



writing, and the like, which require great accuracy and precision in the movement of the muscles brought into action. The dynamometer may be employed for ascertaining the force of the muscular contraction.

Paralysis is of two varieties,—organic and functional. The former is the result of some interruption in the motor tract. The latter is caused by some abnormality in the portions of the cerebral centres which subserve the mental functions. Functional paralysis is usually distinguishable without much difficulty by the facts that there are no indications of trophic changes in muscle, skin, or bone, no alterations of electrical reactions (*vide infra*), no retinal changes, and no diminution of the reflexes, which are indeed often increased. There are, further, usually present other indications of hysteria.

Much more important are the organic paralyses, and some indications for the localisation of these may be useful to the practitioner at this point. These, which are at best only in outline, presuppose in the reader knowledge of what follows in subsequent pages.

*Paralysis from Central Lesion.*—More or less general in distribution, hemiplegic or on both sides, little or no muscular atrophy, deep reflexes usually increased, superficial reflexes increased or rarely diminished, no qualitative change in electrical reactions. With this we may contrast peripheral paralyses, which are usually localised in muscle groups, with marked muscular atrophy, diminution or disappearance of reflexes both superficial and deep, and reaction of degeneration.

Cortical lesions give rise to various monoplegias according to their localisation—the lower central convolutions, facio-lingual monoplegia; the middle, brachial monoplegia; and the paracentral, crural monoplegia. These are usually accompanied by epileptic attacks, anæsthesia, chorea, ataxia, &c.

When the pyramidal tracts are affected in the posterior part of the internal capsule we have hemiplegia with hemianæsthesia. Affections of the corpora striata and optic thalami may produce hemiplegia. Lesion of the anterior portion of the pons gives

rise to hemiplegia with crossed paralysis of the seventh, fifth, and sixth nerves.

*Paralysis from Lesion of the Medulla Oblongata.*—When the pyramidal tracts are affected we have hemiplegia along with paralysis of tongue, soft palate, upper third of the œsophagus, interference with the heart, and respiration, and sometimes the presence of albumen and sugar in the urine.

*Paralysis from Spinal Lesion.*—When one-half of the cord is affected we have what is called Brown-Séquard's paralysis—i.e., paralysis of the muscles on the affected side of the body and anæsthesia on the opposite side. This is accounted for by the fact that the sensory fibres when they enter the cord cross at once to the opposite side, while the motor fibres decussate in the medulla. When the pyramidal tracts in the cord are alone affected we have a pure spastic paralysis.

Lesion of the anterior cornua gives rise to paralysis and atrophy of corresponding groups of muscles, with typical reaction of degeneration, but without any alteration of sensation.

*Paralysis from Lesion of Peripheral Nerves.*—Such lesion produces symptoms resembling those caused by affections of the anterior horns, with, in addition, interference with sensibility.

*Paralysis from Lesions of Muscles.*—In these cases we meet with marked atrophy (except in pseudo-hypertrophic paralysis), without reaction of degeneration, fibrillary twitching, or interference with sensibility.

#### 4. Abnormal Muscular Movements.

Under this heading is included the whole group of spasms or convulsive movements. The more important of these are as follows:—

(a.) *Clonic and Tonic Spasm.*—Spasm (hyperkinesis) of the voluntary muscles, may be defined as abnormal muscular contraction, the result either of pathological irritation or of a physiological stimulus, to which the resulting contraction is disproportionate. It is of two varieties—tonic and clonic—the

former indicating a condition of muscular contraction (tetanus) which remains of nearly equal intensity for a lengthened period (minutes, hours, or days), while under the latter term (clonic) is understood a condition of rapidly alternating muscular contraction and relaxation, whereby particular parts of the body are set in motion.

Of clonic spasms the simplest is *tremor*, which varies from the slightest fibrillary twitching of the lips or tongue to the most well-marked shaking of the limbs which paralysis agitans exhibits. Still more pronounced is the clonic spasm, which occurs in convulsions of all kinds (epileptic, uræmic, hysterical, &c.), in which the whole body may be violently tossed about by the muscular contractions.

Tonic spasm is most commonly seen as "cramp," continuous and painful contraction of muscles individually or in groups; also in catalepsy and in contracture (persistent shortening of muscle, owing frequently to changes in nutrition), and occasionally in the muscles used in certain co-ordinated movements, such as in *writer's cramp*.

In connection with spasm, it is to be noted that certain points are often to be found, pressure upon which either excites or arrests the spasm (motor exciting and motor arresting pressure points). This is particularly noticeable in connection with facial spasm.

A peculiar variety of tonic spasm is seen in *Thomsen's Disease*, the characteristic feature of which is that the patient is unable immediately to make a muscular movement, the muscles in question being thrown into a state of slight tonic spasm. Once, however, the movement commences the spasm disappears.

(b.) *Choreic movements*.—The movements which are characteristic of chorea are well marked. They consist in sudden and irregular contractions of the muscles of the face and limbs which prevent the patient from resting when awake, and if severe may render sleep impossible. The brow wrinkles, the tongue is darted out, the mouth pulled to one side or the other,

the head is twisted suddenly, the shoulders shrugged, hands and feet thrown about irregularly.

(c.) *Athetosis*.—This rare disorder is characterised by slow regular movements of the hands and feet. The fingers are slowly stretched out one after another and then slowly flexed, the hands twisted, the toes extended and flexed. Sometimes similar movements are observed in connection with the head. Athetosis is often unilateral.

(d.) *Ataxic movements*.—These will be subsequently referred to.

### 5. *Mechanical Irritability.*

Over healthy muscles a blow of some force causes a local contraction of sufficient volume to show a swelling under the skin. In various diseased conditions (particularly phthisis) this irritability is so much increased that the slightest tap is followed by such contraction. This is of little or no diagnostic value, as it only indicates muscular exhaustion. The same phenomenon can be observed during the first hours after death.

Tapping over a motor nerve where it runs superficially also gives rise to sudden contraction in the muscles supplied by that nerve. This irritability is often much increased. It usually goes hand-in-hand with the electrical reactions.

### 6. *Reflex irritability of Muscles.*

One of the most valuable diagnostic signs we possess in connection with the nervous system consists in the reflex movements of the muscles. These reflex movements may be excited by stimulation of the skin or mucous membrane (superficial reflexes), or by that of the tendons, facia, or periosteum (deep reflexes), or finally, may consist in the changes of the pupil caused by light, &c.

The value to the physician of these phenomena consists in the fact that their presence or absence gives important indications regarding the integrity of the reflex loop by means of which each individual movement is brought about, and in particular the state of the spinal cord at that level.

*Superficial Reflexes.*

These are very numerous. They include the closure of the eyelids when an object is brought suddenly towards the eye, sneezing when the nostril is irritated, contraction of the soft palate and œsophagus on stimulation of these parts, and coughing when the laryngeal mucous membrane (or sometimes the ear) is irritated.

Those which relate more particularly to the spinal centres are the following :—

(1.) *Plantar Reflex.*—Tickling the skin of the sole gives rise to contraction of the muscles of the foot ; centre in the lower part of the lumbar enlargement.

(2.) *Gluteal Reflex.*—Tickling the skin of the buttock determines in many persons a contraction of the gluteal muscles ; centre probably at the level of the 4th or 5th lumbar nerves.

(3.) *Cremasteric Reflex.*—Tickling of the skin on the inner aspect of the thigh is followed by drawing up of the testicle ; centre at the level of the 1st and 2nd lumbar nerves.

(4.) *Abdominal Reflex.*—On stroking the skin of the abdomen from the costal margins towards the iliac crests, the abdominal muscles contract ; centres lie between the 8th and the 12th dorsal nerves.

(5.) *Epigastric Reflex.*—Tickling the skin of the chest over the 4th, 5th, and 6th intercostal spaces, causes a dimpling of the epigastrium ; centres from the level of the 4th to the 6th or 7th dorsal nerves. The same region of the cord contains centres for the reflex contraction of the erectores spinæ, which occurs when the skin is stroked from the angle of the scapula down to the iliac crest.

(6.) *Scapular Reflex.*—Tickling of the skin in the interscapular region gives rise to contraction of the scapular muscles ; centre at the level of the lower two or three cervical, and the upper two or three dorsal nerves.

*Diagnostic value of the Superficial Reflexes.*—These reflexes are increased—(1) where the cerebral restraining influences are

removed, as sometimes happens in cases of central paralysis and transverse myelitis; and (2) where the grey substance of the cord is unusually excitable, as in cases of strychnine poisoning, tetanus, &c. Diminution or absence of the superficial reflexes results either from interference with the integrity of the reflex loop in question (disease of nerves, spinal nerve roots, white or grey substance of the cord) or from increase in the cerebral inhibitory influence. This latter condition arises in cases of cerebral mischief where there is irritation, and is often of value in distinguishing organic from purely functional paralysis.

### *Deep Reflexes.*

These are produced by tapping over, or quickly rendering tense, tendons or fasciæ.\* The chief of these are—

(1.) *The Knee Jerk.*—If the knee be flexed, and the leg be allowed to hang down loosely, a tap over the patellar tendon will give rise to a contraction of the quadriceps femoris and a consequent jerk forwards of the leg. It is questionable whether this phenomenon can be regarded as a true reflex, but that it is dependent on the state of the reflex loop is evident from the fact that any lesion which affects that loop abolishes the knee jerk. It is occasionally absent in health, about one per cent. of individuals not exhibiting it.

(2.) *Ankle Clonus.*—In certain nervous conditions, a sudden pressure upon the sole of the foot which stretches the muscles of the calf, gives rise to a series of spasmodic contractions of these muscles which recur with great regularity, usually about 5·7 times per second. The clonus can also sometimes be excited by a tap on the muscles on the front of the leg. Similar phenomena may be obtained in the arm.

*Diagnostic value of the Deep Reflexes.*—The deep reflexes are increased—(1) In cases where there is increased excitability of the grey substance of the cord, as in strychnine poisoning and tetanus; and (2) where the inhibitory cerebral influence is with-

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\* Also by tapping over periosteum.

drawn. By far the most common cause is disease of the lateral columns of the cord.

Abolition of the deep reflexes occurs when the reflex loop is interrupted. Any lesion, therefore, which affects the sensory nerves, the posterior roots, the postero-external columns, the grey matter, the anterior roots, or the motor nerves, will produce this effect. These reflexes are thus lost in such diseases as neuritis, poliomyelitis, anterior acuta, progressive muscular atrophy, and disseminated sclerosis. Practically, however, the most important point is that the knee reflex disappears in locomotor ataxia, and that at an early stage of the disease.

### 7. *Electro-diagnosis.*

*Electric Currents* are of the utmost use in diagnosis, but the limits of this work prevent the description of the various forms of apparatus—batteries, electrodes, galvanometers, &c. For such information the reader is referred to special works on the subject.\* It will be sufficient here to indicate very briefly the inferences to be drawn from the information so obtained.

In using electric currents it is important to limit the effects as far as possible to individual muscles or nerves going from one to another and comparing the results obtained. In diagnosis the polar method should always be employed, which consists in placing the pole of the battery, the action of which it is wished to determine, at the point to be stimulated, while the other electrode is placed at some distant part of the body, usually the sternum. Both electrodes, as well as the skin to which they are applied, must be thoroughly moistened. Proceeding in this way we may stimulate either the trunk of a motor nerve, which will cause contraction of all the muscles it supplies, or we may irritate the muscle itself. In acting on the muscle the electrode may be applied either (1) over the *motor point*—i.e., the point at which the motor nerve branch

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\* Such as Tibbit's "Medical Electricity," or Ziemssen's "Elektricität in der Medicin," and Hughes Bennett's "Electro-diagnosis."

enters the muscle, when the muscle as a whole will contract, or (2) the stimulus may be applied to the muscular fibre itself, when there will follow contraction only of the part of the muscle irritated. In order then to be able to make an intelligent use of electric currents in diagnosis it is necessary to know where the motor nerves lie sufficiently superficially to be affected by the current, as well as the position of the motor points of the muscles over the body generally. In figs. 49-55, which are derived from Von Ziemssen's work, the more important of these are given.

The degree of resistance offered by the skin to the passage of electric currents varies. In cases of abnormally high resistance the muscular response is weakened, and, therefore, to prevent error, it is needful to determine the amount of resistance. This is done by means of a galvanometer.

The *Faradic* or *Induced Current* excites muscular contraction when the stimulus is applied to the motor nerve in its course, or over the muscle itself. The contraction so induced varies from a scarcely perceptible change up to tetanus, according to the strength of current used.

The *Galvanic* or *Continuous Current* only gives rise to contraction when the current is opened or closed, not when it is passing. The reaction of each pole should be separately investigated, the other being placed upon the sternum.

The *Law of Normal Contraction* is as follows:—

*Weak Currents*—

Positive pole (anode)—No contraction.

Negative pole (cathode)—Contraction when the current is closed, expressed by the formula C.C.C. (cathodal closing contraction); none when it is opened.

*Currents of Medium Strength*—

Positive pole—Slight contraction both on opening and on closing the current, expressed by the formulæ A.O.c. and A.C.c., anodal opening contraction and anodal closing contraction,—the small c. indicating that the contraction is slight.



Negative pole—Strong contraction on closing the current, expressed by the formula C.C.C', cathodal closing contraction,—the accent on the last C' indicating that the contraction is strong.

*Strong Currents—*

Positive pole—Contraction both on opening and on closing the current expressed by the formulæ A.O.C., A.C.C.

Negative pole—Tetanus when the current is closed, slight contraction when it is opened, expressed by the formulæ C.C.Tc., cathodal closing tetanus, and C.O.c., cathodal opening slight contraction.

Various forms of paralysis may be accurately classified, as Erb has shown, by means of the electrical reactions of the muscles and nerves, as follows:—

a. *No Change in the Electric Excitability* with either form of current (cerebral paralysis before secondary degeneration occurs, and paralysis from disease of the white substance of the cord).

b. *Quantitative Change in the Electric Excitability—*

(α.) *Increase.*—This condition is uncommon, but is sometimes found in the first stage of cerebral hemiplegia, hemichorea, progressive muscular atrophy, peripheral paralysis, and in certain forms of locomotor ataxia, and rarely and transitorily in peripheral paralysis at its commencement. It is characteristic of Tetany.

(β.) *Diminution* occurs in the later stages of such cerebral and spinal paralyses as are accompanied with muscular atrophy. It is met with in pseudo-hypertrophic paralysis, hereditary muscular atrophy, multiple sclerosis, myelitis, &c. It also occurs in muscles which have been paralysed from inaction.

c. *Quantitative and Qualitative Changes* ("Reaction of Degeneration,"—Erb.)—These changes are much more important than the merely quantitative. The reactions of nerve and muscle are different, and must be separately stated as follows:—

*Nerves.*

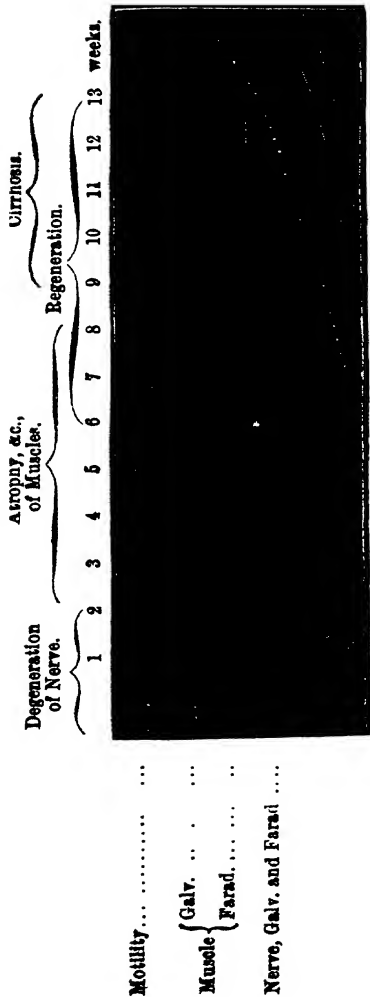
Two or three days after the paralysis has begun, the excitability of the nerves to both currents diminishes, and gradually becomes completely lost. Should recovery take place the excitability reappears, but commonly it is later of being regained than voluntary motion.

*Muscles.*

To the Faradic current they behave much as the nerves do—the excitability being gradually lost, and as gradually regained on recovery taking place.

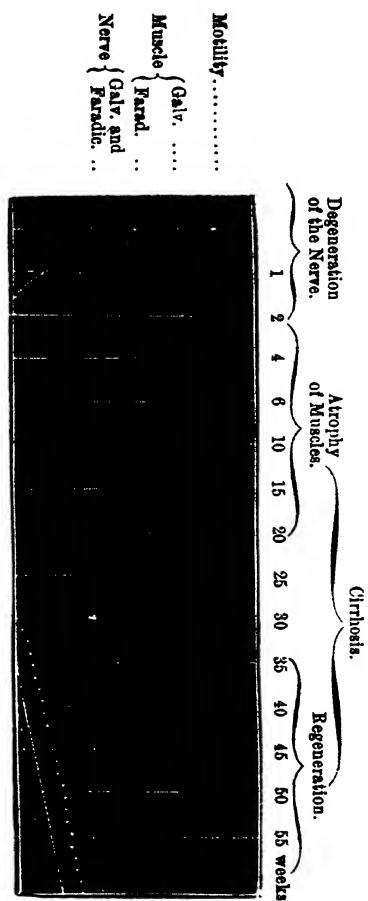
The galvanic excitability falls parallel with the Faradic for about a week, but in the course of the second week it begins to rise, until a point is reached when the muscles contract with stimuli which would have no apparent effect upon them in their normal condition. The muscular contraction thus induced is, however, slow and delayed instead of instantaneous as in health. A qualitative change has also taken place, the positive closing contraction increasing until it equals or surpasses in intensity the negative closing contraction, while the negative opening contraction becomes equal to or exceeds the positive. By comparing the normal law of contraction it will be seen that in this form of paralysis the conditions are exactly reversed. After a time this galvanic excitability of the muscles diminishes, and in incurable cases disappears; but when recovery takes place, the normal state of matters becomes gradually restored. In the accompanying diagrams, constructed by Erb, the electrical reactions in two of these forms of paralysis are graphically indicated. The two selected for illustration show the differences between rapid and slow recovery.

The clinical significance of this “reaction of degeneration” is as follows. It is typical of all forms of peripheral paralysis—traumatic, rheumatic, diphtheritic, or neuritis in any form. It is further met with in cases affecting the grey matter of the anterior horns of the cord, and the corresponding nuclei of the medulla, polio-myelitis anterior, progressive muscular atrophy and bulbar paralysis. The reaction of degeneration is also



\* Return of voluntary motor power.

FIG. 57.—Reaction of degeneration in mild peripheral lesion, with rapid recovery.—(ERR.)



\* Return of voluntary motor power.

FIG. 58.—Reaction of degeneration in severe peripheral lesion, with slow recovery.—(EHR.)

found sometimes in cases of acute myelitis affecting the anterior cornua, and in hæmomyelia. It is characteristic of lead-poisoning. Finally, it is important to bear in mind that the reaction of degeneration is not met with in cases of paralysis from cerebral causes, in hysterical paralysis, or in paralysis due to muscular affections.

It may be useful to give here a brief *resumé* of the electrical reactions in special forms of paralysis—cerebral, spinal, and peripheral.

(1.) *Paralysis from cerebral disease.*—When paralysis results from disease of brain, the electrical reactions are normal in most cases. There is sometimes, however, a slight *quantitative* increase of response which may be due either to irritation of the centres, or to removal of the cerebral inhibitory influence, and at other times a slight *diminution* of response when the muscles have become wasted from disease.

(2.) *Paralysis from disease of the white substance of the cord.*—As a rule in such cases the electrical reactions are normal. If, however, the upper part of the cord be so diseased as to cut off the inhibition of the brain, and at the same time the lower part remain healthy, there will be quantitative *increase*, and the same result follows acute inflammation of the cord from the irritation thereby occasioned. In long standing cases when the muscles have become atrophied, there may be simple quantitative diminution in the response.

(3.) *Paralysis from disease of the grey substance of the cord.*—In such cases we meet with the reaction of degeneration, already described. The area over which the muscles and nerves are so affected depends upon the extent of the cord involved.

(4.) *Paralysis from disease of the peripheral nerves.*—In slight cases the electric reactions are normal. In severe forms there is a total loss of response when either the galvanic or the Faradic current is applied to the nerve, and when the Faradic current is applied to the muscle. The galvanic current acting upon the muscle gives the reaction of degeneration.

(5.) There are besides certain forms of paralysis which cannot as yet be classified under any of these heads, such as *hysterical paralysis* in which the reactions are normal, and *Saturnine paralysis* which shows the typical reaction of degeneration.

### III.—AFFECTIONS OF CO-ORDINATION.

Such complex muscular adjustments as are involved in standing, walking, &c., are probably co-ordinated in the cerebellum and higher nervous centres. They may be interfered with by any affection of the brain, medulla oblongata, spinal cord, or peripheral apparatus.

*Labyrinthine Vertigo*.—In Ménière's disease (a disease of the semi-circular canals) inco-ordination of the muscular adjustments of the body may be observed leading to vertigo or to peculiar movements in particular directions, due to the fact that impressions of the position of the head, derived from the terminal nerve apparatus of the semi-circular canals, have failed or have become erroneous owing to disease of these canals. In other affections of the labyrinth this symptom is met with.

*Ataxia* is a form of inco-ordination much associated with disease of the spinal cord. It is most seen in the actions of standing and walking. The muscles do not move in harmony, the movements made being violent, jerky, and ill directed. The heel is brought down to the ground suddenly and forcibly. The difficulty of movement is much increased when the eyes are shut, so that it is common to make such a patient stand with his heels together and with his eyes closed, in order to test his power of co-ordination. The ataxia then betrays itself in swaying and tottering movements.

*Cerebellar Inco-ordination*.—Affections of the cerebellum and neighbouring ganglia may give rise to various symptoms of inco-ordination—reeling, vertigo, rigidity, and even sometimes to enforced movements, in which automatic movements of the voluntary muscles are preferred in spite of the will.

Loss of muscular co-ordination in connection with speech will be subsequently considered.

It is not at present clear to what this ataxia is in each case due. In many instances, at all events, it results from a lesion of the centripetal apparatus, by means of which the centres are made aware, as it were, of the position of the body. The centripetal paths are various. From the skin, muscles, tendons, joints, semi-circular canals, eyes, &c., such sensory impulses proceed, and anæsthesia of any of these leads to more or less ataxia. But there are some cases of ataxia in which none of these paths are at fault, and then it seems probable that the lesion lies in the centres between the centripetal and the centrifugal fibres. It is also very generally supposed that lesions of the centrifugal fibres occasionally produce ataxia (motor ataxia), and, finally, it is not improbable that an altered tonus of the muscles due to affection of the grey substance of the cord, may be the determining cause.

### C.—Vaso-motor Functions.

As an index of the state of these functions we have to take the condition of the skin as regards palor or redness, temperature, and the amount of the various secretions. Such changes in the tissues as sloughing, &c., are rather to be referred to the trophic nerves.

#### I.—CUTANEOUS VASO-MOTOR AFFECTIONS.

Diffused paleness or redness of the skin may be seen in persons in perfect health (blushing, &c.), but are often associated with nervous disorders such as epilepsy and hysteria, and may be induced by various drugs, as, for example, the flushing which follows the inhalation of nitrite of amyl.

In fever the vaso-motor nerves of the skin appear to be in a condition of abnormal irritability. The hot, cold, and sweating stages of ague seem to depend upon general tonic contraction

of the vessels of the skin, followed by general relaxation, originating in all probability from the centre in the medulla.

More local changes may be brought on by mechanical or chemical irritation, but sometimes occur independently of such, as, for example, the local and circumscribed vaso-motor epileptic aura, which Nothnagel has described. Further, in various neuroses (epilepsy, Graves' disease, &c.), there are to be found scattered over the skin of chest and abdomen red blotches of congestion, to which Trousseau gave the name of *tâches cerebrales*, and which may sometimes be excited by drawing a pencil point over the skin. Various affections of the central nervous system are followed by vaso-motor changes in the skin. In paraplegia the temperature of the paralysed limbs frequently undergoes an increase, which is followed by a diminution when the peripheral nerves are affected. In cases of cerebral hemiplegia the temperature in the paralysed parts is almost invariably slightly elevated, and remains so for some time. Ultimately, however, it falls again, and in old standing cases not only is the temperature on the affected side lower than that on the healthy, but the pulse is smaller and more compressible, and the hand and foot pale and cold. Very high temperatures are sometimes met with in hysterical cases. Unilateral sweating and other vaso-motor disturbances are not uncommon in Graves' disease, epilepsy, and hysteria.

*Visceral Vaso-motor Affections.*—Lesion of the brain and medulla, and even passing psychical disturbances, often determine vaso-motor changes in internal organs (congestion of viscera in hemiplegia, disorders of menstruation from emotions, &c.). The secretions are often affected from such causes. The urine, in particular, is liable to well-marked quantitative and qualitative changes; injury of the 4th ventricle and other areas in medulla, cerebellum, and cord, giving rise to polyuria, albuminuria, and glycosuria. It seems also probable that certain forms of enlargement of the liver and spleen are dependent upon vaso-motor changes determined by affections of the central nervous system.



## CHAPTER XXX.

### Herbous System—(continued).

#### TROPHIC FUNCTIONS OF THE NERVOUS SYSTEM.

AMIDST a great deal that is uncertain in respect to these functions, there are several well ascertained facts which may be briefly alluded to here. The nutrition of all the tissues appears to be under the control of the nervous system. Even the lower portion (cord, nerves) of that system itself are, as the descending degenerations show, controlled from the higher centres. One or two of the more important of these trophic changes may be mentioned.

I. *Muscular Tropho-neuroses*.—Muscles may atrophy as the result of local causes, or as a consequence of long inaction (such as follows paralysis from cerebral hæmorrhage or embolism). Apart from these causes, when active neurotic atrophy of the muscles occurs, it is due either to disease of the nerve cells in the anterior horns of grey matter in the spinal cord or corresponding regions in the medulla, or to affections of those nerve fibres which connect these cells with the affected muscles. It is in such cases that electrical examination gives the “reaction of degeneration” already described (p. 339), and hence its great importance as a means of diagnosis.

II. *Affections of Bones and Joints*.—In all nervous affections, whether peripheral or central, changes in the nutrition of the bones and joints are liable to occur. Amongst the best known are those which follow locomotor ataxia, which may be acute articular swelling, closely resembling rheumatism,

or chronic degenerative changes, leading to great deformities and strong predisposition to fractures. The important point to note in regard to these affections is that they are unaccompanied with pain.

III. *Affections of the Skin.*—So far as is yet known, the nerve fibres connected with cutaneous nutrition leave the cord, along with the posterior sensory nerves, and it appears probable that they spring from the cells of the posterior horns of the cord. Various eruptions, such as erythema, urticaria, eczema, herpes, may arise as the result of disease of these nervous structures, and Paget has described an affection of the skin of the fingers, "glossy skin," which is due to the same cause. Still more important, as belonging to this category, are the acute and chronic bed-sores, which are so common and so troublesome in spinal cases. And, finally, there have to be noted the occurrence of pigmentation, and of affections of hair, nails, and cutaneous secretory apparatus, and the more profound lesions of lepra anæsthetica, all of which must be included among the cutaneous tropho-neuroses.

IV. *Affections of the Secretory Glands.*—Salivation and lacrymation, as well as the flow of the bile and other secretions, are under the influence of the nervous system, but do not give diagnostic indications further than has been already noted in other parts of this work.

V. *Affections of the Viscera.*—Of these too little is known to aid in diagnosis.

## CHAPTER XXXI.

### Nervous System—(continued).

#### CEREBRAL AND MENTAL FUNCTIONS.

IN many diseases of the nervous system the intellectual powers are affected. The powers of attention and memory of the patient are put to sufficient proof while the physician is informing himself regarding his history. Decadence of the power of judgment may betray itself in connection with the business transactions of the patient, which, if obviously irrational, will usually be communicated by his friends. The most striking interference with the intellectual powers is, however, loss of consciousness, or coma.

*Coma* is met with in simple fainting, in injuries of the head, in apoplexy (in which case it is accompanied with paralysis), in epilepsy (usually accompanied with convulsions), in hysterical attacks, in catalepsy, in uræmia, in severe attacks of fever of various kind, and in narcotic poisoning. It is sometimes very difficult to establish a diagnosis between alcoholic poisoning, for example, and apoplexy. The state of the pupils, the smell of the breath, the condition of the heart, and the presence or absence of paralysis, will, however, usually make clear the nature of the case. *Coma vigil*, in which the patient lies unconscious, with the eyes wide open, is met with in cerebral diseases.

There are certain other disorders of intelligence which frequently occur in mental disease, and which must be noticed here.

*Illusions* are objective disorders of perception—a sound is heard, or an object seen; but both perceptions are misinterpreted.

*Hallucinations*, on the other hand, are subjective disorders of perception. The patient may imagine, for example, that rats are running over the bed-clothes, or that he hears people calling to him, when no foundation exists for either belief. These are hallucinations. But if he observes some dark object on the bed and take it to be a rat, that would be an instance of an illusion.

*Delusions* have no relation to perception. They are purely mental, and are only met with in the insane. It is not uncommon, for example, to meet with such delusions as that the patient believes himself to be the Deity.

*Delirium*, or wandering of the mind, indicated by incoherent speech, may consist in low muttering or in wild and furious shouting. The former variety is most frequently met with in cases of nervous exhaustion, the result it may be of fever, or of any grave organic disease. The more noisy form of delirium occurs in meningitis, in acute mania, and as the result of some poisonous ingredient circulating in the blood, such as alcohol, fever poison, belladonna, carbonic acid, and other substances. Delirium may also be caused by reflex irritation in connection with such organs as the stomach or uterus. A variety is not uncommon in pneumonia, and is even occasionally met with in phthisis pulmonalis.

It is also important to note further the condition of the patient in regard to mental *emotions*, whether these are under full control or not. This is very obviously not the case in hysterical persons, and in many other nervous affections the patient may be observed to be emotional and excitable.

**Speech.**—The physiology and pathology of speech are of a most complex nature, and can only be touched upon here. We must first of all shut out all imperfections of speech due to

laryngeal affections, which are grouped under the head of aphonia, and which are apart from the present subject.

In order that an individual may employ language as a means of communicating with his fellows, he must first of all be able to understand their gestures or speech—the receptive and regulative department of speech; and secondly, he must be able to express his own mental state by means of gestures or speech—the emissive and executive department.

The *receptive* function is performed by means of the various end organs of special sense, and by the nervous apparatus which connects these with the central organs. Persons who are born blind, or become both blind and deaf, are examples of impairment of the receptive function of speech.

The *regulative* function of language, or that whereby the individual reduces to order and to comprehension the impressions which reach his brain, is impaired in the disease known as amnesic aphasia. This affection of speech takes many different forms. Sometimes the patient cannot remember the names of things, while every other part of his sentences comes easily to him. Sometimes he cannot name an object held up in front of him, while he can talk and write fluently. At other times the initial letter of the word may be all that he can recollect. Finally, much more pronounced cases are often met with, in which the patient cannot understand spoken or written language at all, and in which his own words are meaningless. The lesion in cases of amnesic aphasia corresponds to the distribution of the posterior and terminal branches of the middle cerebral artery.

The *emissive* function of language—viz., that by which the speech impulses are co-ordinated before being sent to the executive department, is impaired or lost in the disease known as ataxic aphasia. The patient understands perfectly what is said to him or what he reads, and he has no difficulty in articulation, but he is unable to communicate his thoughts by speech or by writing (agraphia). His speech consists of what have been called “recurring utterances,” which may or

may not chance to be appropriate to the occasion, and which do not possess any intellectual value. The lesion is usually in the third left frontal convolution or in the island of Reil.

The *executive* department of language, that, namely, which regulates articulation, is impaired in diseases of the medulla oblongata (bulbar paralysis), and in general paralysis of the insane. In the former affection the speech is "scanning," that is, the words are uttered syllable by syllable. In the latter the syllables are misplaced, and there is stuttering and stammering.

For details regarding the pathology of the function of language special works must be referred to. Enough has been given here to show how any particular case is to be classified.

**Sleep.**—The disorders of sleep are of considerable practical importance. They are mainly three—

1. *Somnolence*.—Apart from the natural aptitude for sleep possessed by persons of a lethargic temperament, the causes of somnolence are mainly as follows:—Exposure to external cold, especially when combined with insufficient nutriment; overloading the stomach with food; dyspepsia; blood poisoning (uræmia, fevers, poisoning with narcotics, alcohol, carbonic acid, &c.); disease of the brain.

2. *Insomnia*, or want of sleep, may be directly due to pain. It may further arise from excessive mental work, worry, anxiety, from dyspepsia, from the use of tea or coffee, from cerebral disease, from insanity, and from heart disease.

3. *Somnambulism*.—In this case also a definite cause, similar to the above, may usually be found.

## CHAPTER XXXII.

### Herbous System—(continued).

#### CONDITION OF CRANIUM AND SPINE.

**Cranium.**—The condition of the cranium sometimes affords important indications in nervous cases. Thus in epilepsy we may find a distinct localised depression, the result of an old depressed fracture, and which may indicate the cause of the disease. In chronic hydrocephalus, again, the peculiar shape of the skull is globular, the frontal bones prominent, and the eyeballs protruding; the sutures are open, and the fontanelles large and pulsating. Occasionally, also, tumours of the cranial bones may be detected which may have given rise to symptoms of compression.

**Spine.**—Examination of the spine includes inspection, palpation, percussion, and the “hot sponge test.”

**Inspection.**—The patient should, if practicable, be stripped, and be made to stand upright, with the feet close together and firmly planted. If an ink mark be made on the skin over the tip of each spinous process, the line of the spine will be rendered distinct, and any lateral curvature will readily be detected. At the same time, any displacement of the column caused by angular curvature will become apparent.

**Palpation** of the spinal column should be practised both posteriorly and anteriorly through the abdominal walls. Tumours of the vertebræ can thus be detected, as well as the presence of local tenderness at any particular point.

*Percussion* of the spine posteriorly causes pain when there is disease of the vertebræ or of the spinal membranes, in myelitis, in spinal irritation, &c.

*Hot Sponge Test.*—This test consists in passing down the spine a sponge which has been wrung out of warm water, and which is not so hot as to be unpleasant to the healthy skin. In certain cases, particularly in myelitis, pain is experienced by the patient as the sponge passes over the seat of the disease.



## CHAPTER XXXIII.

### Locomotor System.

#### BONES—JOINTS—MUSCLES.

**Bones.**—In cases of suspected syphilis, the surface of the bones, particularly the cranium and tibia, ought to be carefully examined to detect the presence of nodes. The softness of the osseous tissues generally in cases of mollities ossium, and the enlargement of the articular ends of the bones and their altered shape in cases of rickets, are also to be looked for.

**Joints.**—An examination of the joints should be made whenever pain is complained of in them, or when the presence of pyæmia, rheumatism, or gout is suspected. Allusion has already been made to the joint affections which are met with in locomotor ataxia. Hysterical joint affections are more surgical than medical in their nature. The acute swelling and inflammation of the joints in rheumatic fever and in gout is usually very obvious. In rheumatoid arthritis, also, the joints become enlarged, deformed, and stiff, movement being accompanied with distinct crackling.

**Muscles.**—The condition of the muscular system has already been considered under the head of the nervous system.

## APPENDIX.

### METHOD OF PREPARING THE SOLUTION OF NITRATE OF MERCURY USED FOR THE DETERMINATION OF UREA.

THIS standard solution is best prepared from pure mercury. Of this substance 71.48 grammes are to be carefully weighed out, placed in a large beaker-glass, and treated with five times that weight of pure nitric acid of specific gravity 1.425. The solution so obtained is to be warmed in a water bath until all the nitrous acid vapour has been driven off, and the solution has become colourless. A few drops more of the acid are to be added until nitrous fumes cease to develop themselves, the solution evaporated to the consistence of syrup, and then carefully diluted with water up to one litre. The accuracy of the solution of nitrate of mercury so obtained must now be tested by comparing it with a standard solution of urea, prepared in the following manner:—Two grammes of pure urea, dried *in vacuo* over sulphuric acid, are dissolved in water and diluted until the fluid has a volume of exactly 100 c.c. Of this solution 10 c.c. contain 200 milligrammes of urea. By using the volumetric method detailed in the text, the standard solution of urea being substituted for the urine, the exact strength of the solution of nitrate of mercury prepared may be readily ascertained; and it can, if necessary, be altered in concentration by adding more mercury or more water, as the case may be, so as to bring it to the exact strength mentioned in the text—viz., 1 c.c., corresponding to 0.01 grammes of urea.

## APPENDIX B.

### METHOD OF CASE-TAKING IN THE UNIVERSITY CLINICAL WARDS OF THE EDINBURGH ROYAL INFIRMARY.

**NAME—AGE—OCCUPATION—PLACE OF BIRTH—PLACE OF RESIDENCE**  
—DATE OF ADMISSION—DATE OF EXAMINATION—COMPLAINT  
—DURATION OF ILLNESS [*Insert Thermometer*].

**History.**—Hereditary Tendencies—Habits as to Food and Drink—  
General Surroundings at Home and at Work—Previous  
Illnesses and Accidents—Time and Mode of Origin, and  
Course of present illness.

#### **State on Admission.**

**General Facts.**—Condition as to Height and Weight—Develop-  
ment—Muscularity—Obvious Morbid Appearances, as  
Jaundice, Dropsy, Cyanosis—Evidences of Injury or Previous  
Disease—General Appearances and Expression of Face—  
Temperament (if well marked)—Attitude (if unusual)—  
Temperature.

**Alimentary System.**—Lips—Teeth—Gums—Tongue—Secretions  
of Mouth—Fauces—Deglutition—Appetite—Thirst—Sen-  
sations during Fasting—Sensations during and after Eating  
(*Comfort or Discomfort—Pain—Weight—Distension—Heart-  
burn—Nausea*)—Acidity—Flatulence—Eruclation—  
Waterbrash—Vomiting (*Characters, Macroscopic and  
Microscopic, of Vomited Matters*)—State of Bowels and Char-  
acter of Fæces. Abdomen—Inspection (*Prominence—  
Retraction—Distension—Flaccidity*)—Palpation (*of Parieties  
of Contents, normal or abnormal—Tenderness—Fullness—  
Fluctuation*)—Percussion (*Vertical Dullness in Mammillary  
line, and, if necessary, outline of Liver, &c.*)

**Hæmopoietic System.**—Lymphatic Vessels and Glands—Ductless  
Glands (*Spleen—Thyroid*)—Microscopic Characters of Blood  
(*Corpuscle Counting, and Determination of Hæmoglobin if  
necessary.*)

**Circulatory System.**—Subjective Phenomena (*Pain—Palpitation  
—Faintness—Dyspnœa*)—Inspection (*Form and Appearance  
of Præcordia*)—Palpation (*Position and Character of Cardiac  
Impulse*)—Percussion (*Superficial and Deep, Outline if neces-  
sary*)—Auscultation—*Rhythm and Quality of Sounds in  
Mitral, Tricuspid, Aortic, and Pulmonary Areas over Gen-  
eral Surface of Heart and Main Vessels*)—Pulse (*Frequency  
—Rhythm—Character*)—Arteries, Capillaries, and Veins.

**Respiratory System.**—Breathing (*Frequency—Rhythm—Type—  
Painfulness*)—Cough—Sputa (*Macroscopic and Microscopic  
Characters*) Nares—Pharynx—Larynx (*Voice—Pain—Ten-  
derness—Laryngoscopic Examination if necessary*)—Inspec-  
tion (*Form and Action of Thorax, Measurement if necessary*)  
—Palpation (*Vocal Fremitus*)—Percussion (*Anterior and  
Posterior at corresponding points on the two Sides of Chest*)—  
Auscultation (*Determination at each point, during Natural  
and Deep Respiration, of the Duration of Respiratory Sounds,*

*their Quality or Character, Accompaniments or Superadded Sounds, and of the Vocal Resonance).*

**Integumentary System.**—Subjective Phenomena—Skin (*Dryness—Moisture*)—Obesity—Emaciation—Œdema—Emphysema—Eruptions (*Distribution—Elements of Skin involved—Type—Cause*).

**Urinary System.**—Subjective Phenomena (*Pain or Uneasiness in Loins, Bladder, or Urethra*)—Micturition (*frequency*). **Urine.**—Quantity—Colour—Specific Gravity—Chemical Reactions (*Acidity—Alkalinity—Albumen—Sugar—Bile—Amount of Urea if necessary*)—Deposits (*Macroscopic and Microscopic Characters*).

**Reproductive System.**—**Male.**—Subjective Phenomena—Functions—Testicle—Epididymis—Prostate—Urethra. **Female.**—Subjective Phenomena—Catamenia—Abnormal Discharges—Vagina—Uterus (*Examination, Digital, with Speculum and Sound if necessary*)—Ovaries.

**Nervous System.**

**Sensory Functions.**—Sensations (*Pain—Heat—Cold—Formication—Numbness—Tingling*)—Sensibility to Touch—Heat—Tickling—Pain. Muscular Sense. Sight (*Ophthalmoscopic Examination if necessary*). Condition of Pupil. Hearing—(*Otoscopic Examination if necessary*). Taste. Smell.

**Motor Functions.**—Organic Reflex (*Swallowing—Breathing—Micturition—Defæcation, &c*)—Skin Reflex—Tendon Reflex—Voluntary (*Systematic Examinations of Groups of Muscles if necessary*). Co-ordinating—Electric Irritability (*Faradic, Voltaic*).

**Vaso-motor and Nutritive Functions.**—(*Local Congestions—Pallor—Œdema—Inflammation—Sloughing—Wasting—Perspiration, &c.*).

**Cerebral and Mental Functions.**—Intelligence (*Hallucinations—Illusions—Delusions—Torpor—Coma*)—Attention—Memory—Speech (*Comprehension of Language, heard, seen; Utterance of Language, spoken, written*)—Sleep.

**Cranium** (*Peculiarities*)—**Spine** (*Form and Appearance—Percussion—Hot Sponge Test*).

**Locomotor System.**—Bones—Joints (*Pain—Swelling—Effusion—Mobility*)—Muscles (*Rigidity—Flaccidity—Cramp—Twitching, general or fibrillary—Hypertrophy—Atrophy*).

**Provisional Diagnosis.**

**Treatment.**—Medicinal—Dietetic—General Directions.

**Further Reports.**

**In Acute Cases** report Daily, or more frequently if necessary.

**In Chronic Cases** report Once or Twice a-week, always noting any change of Diagnosis or Treatment.

At Conclusion of Case note the result.

[*Signature of Clerk and Initials of Resident Physician.*]



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